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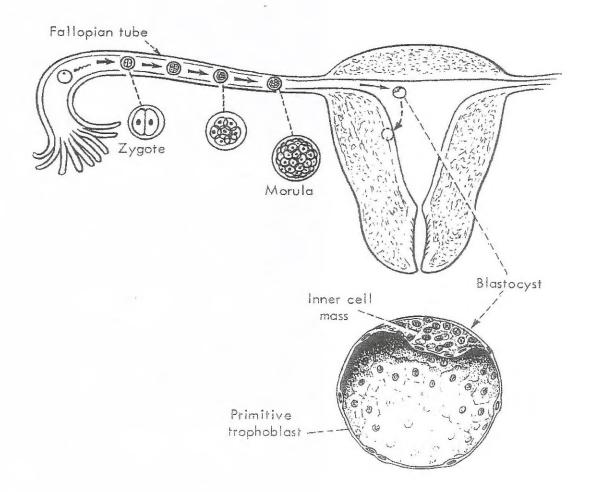
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(Physiology Of

Pregnancy)

Fertilization
Placenta
Maternal adaptation
Diagnosis of pregnancy
Ante-natal care

50 M OVULATION 12th Day FERTILIZATION Spermatozoon Zygote Oestrogen 15 10 25 Day 5 of Morula 🕲 Blastocyst Cycle Embedded Blastocyst 26th Day Repair Decidua Proliferation Secretory



Physiology of pregnancy

Fertilization

- Definition.....The union of a mature ovum & a mature spermatozoon at ampulla of F.tube * (bet. outer & middle 1/3) → zygot
- Transport of sperms no very occurs through the action of tail stimulated by
 Mature sperms 122- 22

- Mature sperms [22x or 22y] reach F.tube.....within 40 min some rap that Capacitation of sperms starts..... within the cx beringe vehanges in sperms to tits ability to fertilize Takes about 26h. Removal of excess proteins, present in acrosomal Cof.

. Production of enzymes e.g. hyaluronidase, " 6 Mageness."

Transport of ovum

- Ovulation \rightarrow completion of meiosis $I \rightarrow 1^{ty}$ oocyte $+ 1^{st}$ polar body

- Ovum pick up occurs by the tubal fimbria → then it is carried by * > passive fluid currents (helped by ciliary action & peristalsis)

The Union

Nickening of 2.P (fe tilization ment).

- 1sperm penetrates the ZP (Polyspermia is prevented by: Zonal block)
- Meiosis II starts after fertilization ()

➤ Sperm head swells * to form the ♂ pronucleus

Differentiation

- Zygot rapidly divides by mitosis...2...4...8 → BLASTOMERES
- A MORULA is formed (round mass ≈ 16 cells)

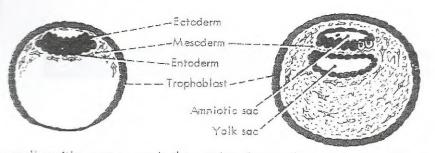
- It reaches the uterine cavity after 3 days from fertilization delayed by

- It is nourished by secretions from the tube (tubal milk) when the sphireter

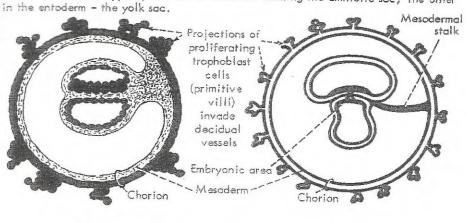
• Fluid will then accumulate → a BLASTOCYST/Charlen).

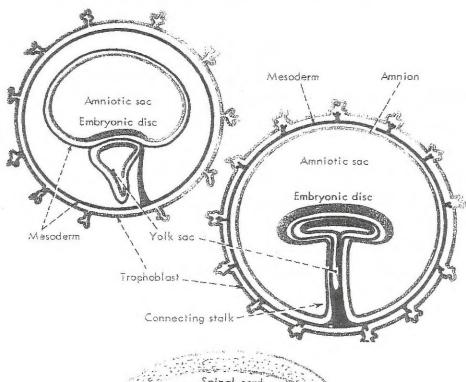
- It remains free for 3 days in uterine cavity

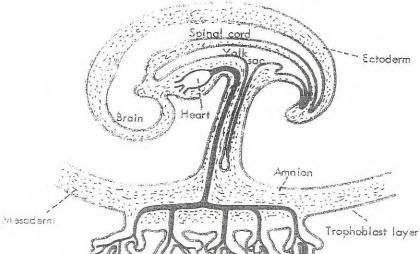
- It is nourished by secretions from the endometrium (uterine milk)
- The blastocyst will be divided into 2 masses:
 - INNER CELL MASS $\rightarrow \approx 100-250$ cells \Rightarrow will form the embryo
 - OUTER CELL MASS → trophoblast ⇒ responsible for nutrition



Two small cavities appear, one in the ectoderm forming the amniotic sac, the other in the entoderm - the volk sac.







Implantation:

- Protection against the invasive power of trophoblast

 | Protection | Invasion | Protection | Pr

> absence of this layer → adherent placenta → failed pl. delivery (pl. accepta)

Formation of the chorionic villi

• The chorionic villus is formed of 2 layers of trophoblast:loss of Coll - Cytotrophoblast = Langhan layer.....inner idnove of Syncitiotrophoblast outer

- Chorionic villi are 2 types
 - Anchoring → Fixation & attachment

.Solid finger like projections into the decidua → 1° villi .Mesoderm develops in the 1^{ry} villi (CT core) \rightarrow 2^{ry} villi . Vascularization of 2^{ry} villi with fetal vessels $\rightarrow 3^{ry}$ villi

→ @ Food & nutrition Branches of Anchoring will. The trophoblast invades the endometrial vessels (pirela.) ->

1' wave of invasion Form Charle-diciolard space).

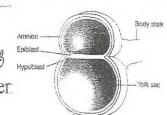
At 20 weeks trophoblast invades media of spiral compatibility of spiral evessels -> 2" wave of invasion

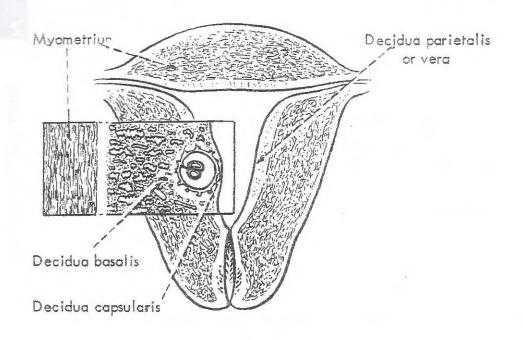
Tunctional unit "(Endecrinal funct.).

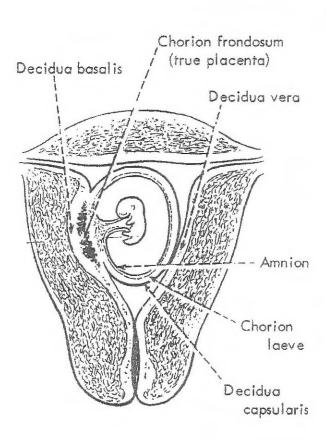
.HCG → maintenance of CL to produce 'P' for 7-10 wks till the placenta is formed

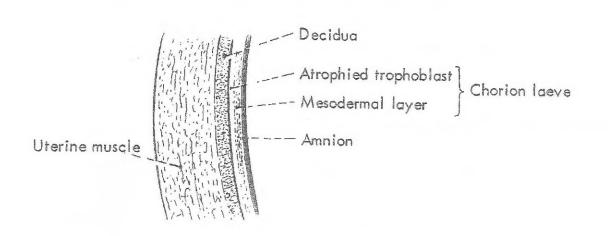
Early development of Embryo (till 7 wks; after this \rightarrow a fetus *)

- 7^{th} day \rightarrow two layers (endoderm and ectoderm)
- 10^{th} day \rightarrow amniotic cavity and primitive yolk sac are formed \mathcal{B}
- 16th day → three layered embryo (endoderm, mesoderm, ectoder.









Placenta

Normal structure

>	Shape.			4			,	4				P	,	,		+	discoid
---	--------	--	--	---	--	--	---	---	--	--	--	---	---	---	--	---	---------

- ➤ Weight......500 gm **
- ➤ Site.....UUS (60% posterior) → site of implantation
- ▶ Thickness......2.5 cm in center → gradually thins towards periphery
- ➤ Cord Insertion....eccentric ^a

Placental formation

- > Two surfaces
 - FETAL SURFACE is smooth & covered by amnion "
 - 2 MATERNAL SURFACE is divided into 15-20 cotyledons (lobes)
- ➤ Two parts "
 - FETAL PART = Chorion frondosum (chorionic plate) =

Trophoblast + mesoderm + fetal vessels projecting as villi into the intervillous space

Chorion leave

 ζ is the rest of chorion not sharing in placental formation

② MATERNAL PART ➡ <u>Decidua basalis</u> (decidual / basal plate)

Before 12 veeks - sureus
15 a pelvic organ.
Afte 12 ... - surteus
is apelvi-aldonimal

Decidua capsularis

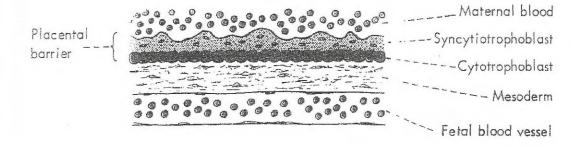
 ζ is the rest of the decidua overlying the developing ovum Decidua parietalis (vera)

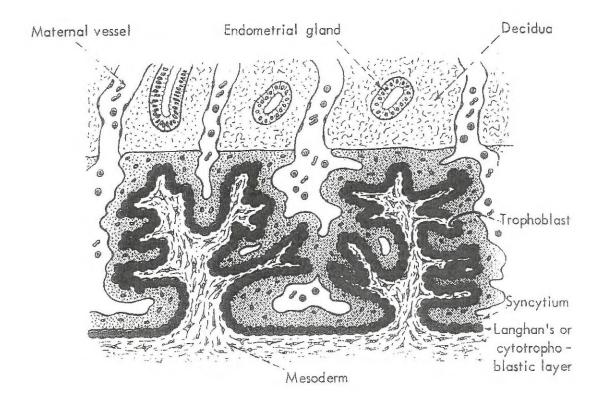
 ζ is the decidua covering the rest of the uterine cavity

> Fusion of D. capsularis & D. parietalis occurs at 12 wks obliteration of the whole uterine cavity by the fetus

Fetal membranes (could be separated)

The Amnion	The Chorion			
-The inner membrane	-The outer membrane			
- Covers the fetal surface of the placenta & the cord	- In contact with the uterine wall, it ends at the margin of the placenta			
-Transparent, glistening	-Less transparent			





Functions of Placenta $\Phi\Phi$

- Mechanical attachment
 - A Placental barrier:- "
 - 1- Cytotrophoblast 2- Syncitiotrophoblast (↓ at 5th 6th month)
 - 4- Fetal capillary endoth. + its basement memb. 3- Mesoderm

*The placenta becomes thinner as pregnancy advances *

- *The placental is permeable to many drugs & organisms e.g.
 - Drugs reg oral anticoagulants & oral hypoglycemics
 - Bacteria TB, syphilis, malaria, toxoplasma
 - Viruses MMR, CMV, chickenpox, polio
- - > Nutrition *
- Water & electrolytes..... simple diffusion
- excretion Glucose, amino acids.........facilitated diffusion
 - Ca, Fe, minerals.....active transport
 - Immunoglobulins & LDL.....pinocytosis
- - ▼ Hormone production ⇒ sex steroids (estrogen & progesterone)

Source \Rightarrow < 7 weeks \rightarrow from CL mainly

7-10 weeks → from CL+ placenta (syncitium ^a)

> 10 weeks → from placenta mainly

Function \Rightarrow responsible for all changes in preg Level = they continue to rise till end of preg

Feto-placental unit:

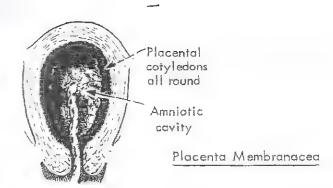
- Placenta can convert cholesterol (C_{27}) into progesterone (C_{21})
- Placenta can convert androgens (C₁₉) into estrogens (C₁₈)
- However, placenta can't convert progesterone into androgens : it must be supplied with androgens first e.g.:- DHEA & DHEA-S from both maternal & fetal sources (suprarenal gland mainly)

∴ ↓ in anencephaly **

Estrogen Level

- Estrone (E₁), Estradiol (E₂) $\rightarrow \hat{\mathbf{T}}$ 100 times
- Estriol $(\mathfrak{E}_3) \to \widehat{\mathfrak{U}}$ 1.000 times (the index " of feto-maternal unit)
- Estetrol $(E_4) \rightarrow \text{only formed in preg (of little significance)}$

Progestarone Level > 50-150 ug/ml (10 times luteal level)

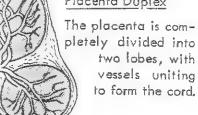


Placenta Bipartita

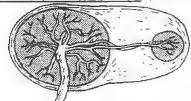


The placenta is partly divided into two lobes, with connecting vessels.

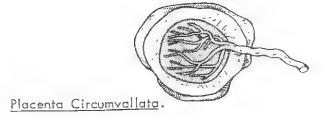
Placenta Duplex



Placenta Succenturiata ('substitute')



(A variant of Duplex)



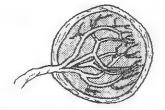
Uterine wall Placental tissue

Attachment of membranes to fetal surface Reduplicated and infarcted chorion



Placenta Fenestrata

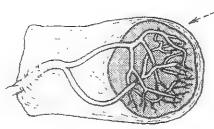
A defective area appears in the middle of the placenta. It may be wrongly taken for the site of a missing lobe.



Battledore Placenta

Sometimes the cord has a marginal instead of a central insertion. This has no clinical significance.





Abnormalities of Placenta

 Z_{1} \mathfrak{q}

> Shape

- 1. Bipartite placenta = 2 equal lobes connected by.....membranes
- 2. Bilobate placenta => 2 equal lobes connected by..... placental tissue
- 3. Placenta fenestrata a window is present (a part of placenta is missed)

4. Placenta succenturiata (succenturiate lobe / lobes)

- Small accessory cotyledon/s attached to placenta by membranes
- May be torn away during delivery \rightarrow retention \rightarrow PPHge $^{\text{m}}$ or p.sepsis
- Diagnosed by routine examination of pl. ightarrow site of torn vessels on margin

5. Placenta circumvallate (extrachorial placentation)

- The chorionic plate (ch.frondosum) is < the basal plate (D.basalis)
- The fetal margin shows a white ring formed of decidua
- May lead to abortion......CFMF, IUGR, PTL, IUFD......accidental hge

6. Placenta membranacea

- The chorion leave does not atrophy → large thin placenta (15–20 inches)
- May lead to placenta previa
- If accompanied by vasa previa → APHge of fetal origin ✓

> Site

- o In LUS → placenta previa
- o On septum → liability to abortion, APHge, PPHge or retention
- o Elsewhere (as tubes or peritoneum) → ectopic pregnancy

> Size

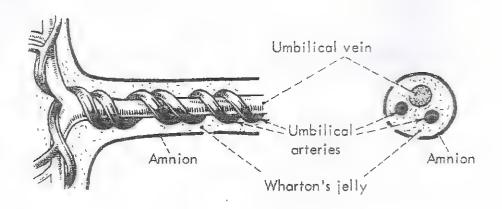
- Small (associated with IUGR or infarcts) = placental insufficiency
- o Large (hyperplacentosis) = syphilis, Rh, DM, twins, placenta membranacea
 - . Syphilis: large, pale, friable / Endarteritis obliterans / Spirochetes
 - . Rh isoimmunization: large, pale, edematous

Abnormal adherence (absent Nitabuch layer)

- P. accreta → placenta reaches basal layers of decidua, may reach muscle
- P. increta → penetrates muscle but does not reach serosal surface
- P. percreta → perforates uterus
- ▶ Placenta infarctions → esp. in PET due to narrow blood v. → red infarcts
 Normal calcification (physiological) → white infarcts

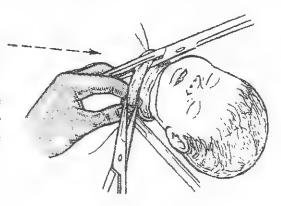
> Tumors of placenta

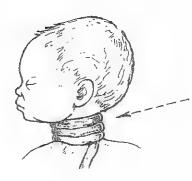
- o Placental polyp → retained parts of placenta after delivery
- o Vesicular mole & Choriocarcinoma
- Chorioangioma → vascular malformation (tumor) → polyhydramnios
- Abnormal attachment of the cord......



Cord Round the Neck

One or two loops of cord are quite often seen round the baby's neck at vertex delivery and normally do no harm. As soon as the neck is visible at the vulva the loop should be clamped and divided before delivery of the shoulders and trunk.





Much less frequently six or seven loops are drawn tightly round the neck. As the fetus descends the cord tightens, the blood supply is interrupted and the baby is stillborn. This is one cause of sudden acute fetal distress.



Single Umbilical Artery

This abnormality is frequently associated with other congenital abnormalities in the fetus.





Umbilical Cord [funis]

Structure

- Length → about 50 cm
- Diameter → about 2 cm
- Contents → 2 arteries (non-O₂) & 1 vein (O₂) * carrying <u>fetal blood</u> along with remnants of allantois in myxomatous tissue (Wharton's jell). Vessels are convoluted (length of vessels > cord)
- The amniotic membrane covers the umbilical cord *

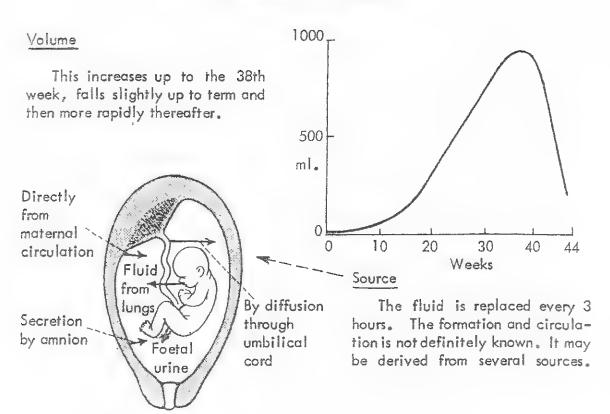
One vein carries oxygenated blood to the fetus
Two arteries carry reduced blood from the fetus to placenta

Ponormalities

- 1. Length
 - ➤ Very long (>100 cm) may lead to:
 - Coiling around fetus
 - True knots
 - Cord presentation & prolapse **
 - ➤ Short cord (<32 cm) may lead to:
 - Failure of . engagement & descent of fetus
 - presentation (malpresentation e.g. transverse lie)
 - external cephalic version or forceps
 - Fetal asphyxia (distress) or rupture of cord or
 - APHge (accidental hge)
 - . Uterine inversion
- 2. Abnormal attachment: may be:
 - ➤ Central
 - ➤ Marginal (battledore) **
 - Velamentous insertion of the cord
 - Vessels are inserted into the membranes (& not placenta)
 - If the traversing vessels pass below the presenting part in the region of the cervix they are

 called.....∨asa previa
 - It is usually associated with placenta membranacea
- 3. Knots in cord, may be:
 - * True ⇒ fetus passes through loops of the cord → may lead to fetal distress
 - * False \Rightarrow localized varicosity in a collection of Wharton's jelly \rightarrow no effect
- 4. Congenital umbilical hernia
- 5. Tumors / custs (as myxoma and sarcoma)
- 6. Absence of one umbilical artery
 - * Common in DM
 - * May be associated with CFMF, IUGR, prematurity

AMNIOTIC FLUID



Amniotic fluid

- Layers of amnion (0.5 mm → 5 layers)
 - 1. Cuboidal epithelium
 - 2. Basement membrane
 - 3. Compact layer (reticular fibers arranged in bundles)
 - 4. Fibroplastic layer
 - 5. Spongy layer (contains mucous \rightarrow can glide upon chorion)
- > Source of omnion.....amniogenic cells (from fetal ectoderm)
- > Source of amniotic fluid
 - 1. Maternal => transudation (esp 1st trimester) through placenta & cord
 - 2. Fetal Urine // (esp in the 2nd half)
 - Lungs (bronchial, buccal & salivary glands)
 - Amniotic epithelium
 - Transudation from fetal skin & umbilical cord
 - → Then it is removed by transudation + fetal swallowing i.e. dynamic circulation **

> Volume

$6 \text{ wk} \rightarrow 5 \text{ ml}$	10 wk → 30 ml	$20 \text{ wk} \rightarrow 300 \text{ ml}$
$30 \text{ wk} \rightarrow 600 \text{ ml}$	$36 \text{ wk} \rightarrow 1.000 \text{ ml}$	$38-40 \text{ wk} \rightarrow 800 \text{ ml}$

➤ Composition

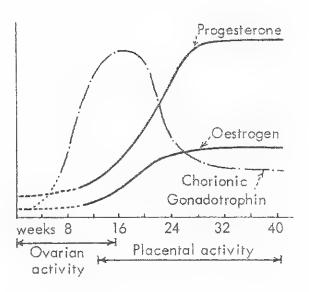
- 99 % clear watery
- 1-2% crystalloids & colloids [+ vernix caseosa, desquamated epith., lanugo hair]
 - CHO (glucose & fructose) proteins (albumin & globulin) lipids
 - Hormones (E.& Pr.) electrolytes (Na. K. cl. Cu.)
- Physical properties
 - Colorless → later on will be turbid
 - Specific gravity \rightarrow 1010-1020
 - Reaction → slightly alkaline ^a (7-7.5)

➤ Functions Φ

1. During ρ	2. During labor	
.Protection against trauma	. Helps cervical dilatation	
.Prevents adhesions bet.	. Allow free movement	. Prevents direct fetal &
	of fetus → muscular	placental compression
	development	by the uterine wall
.Prevents infection	. Development of alveoli	. Wash birth canal after ROM

Abnormalities

- o Volume (û...polyhydramnios, √....oligohydramnios, PROM)
- Meconium staining
 meconium aspiration syndrome
- o Inflammation chorioamnionitis
- o Amniotic cysts......bands (may lead to amputations)



ंत्रकार्यकार्यकार्यः	lacental hormones
Hormone	Role
Human chorlonic gonadotrophin (hCG)	Initially maintains the corpus luteum's secretion of progesterone and cestrogen; later it may have a role in regulating placental cestrogen secretion and in modulating the maternal immune response
Cestrogen	Over 90% is in the form of oestriot; it is involved in uterine growth, cervical changes, and breast development
Progesterone	Smooth muscle relaxation, acting on the uterus, gastrointestinal tract and ureters. Also has a role in regulating maternal physiological changes
Human placental lactogen (hPL)	Mobilizes maternal free fatty acids, improving glucose availability for the fetus

Hormones

- * Steroids → estrogen & progest......from CL & placenta *
- Proteins → HCG & HPLfrom syncitio-trophoblast

1) Human chorionic gonadotrophin

- ▶ Sime of production (a glycoprotein) "
 - Appears at 1st day of implantation
 - Can be detected within 10 days of fertilization (conception) a i.e. before missing a period

> Level

- It û rapidly in early pregnancy → level doubles every 2 days
- Reaches a peak at 70 day (10 wk) gestation (≈ 50.000 mIU/mI) x
- Then it \$\Partial \text{ at 100 d (14 wk) \$\approx (5.000) & remains as such till term \$\int \text{Formation of \$\ell\$. \$\sigmathsquare \text{fordisc.} \$\ell \text{7 \in } \text{\$\sigmathsquare}\$ of \$\ell\$. \$\sigmathsquare \text{\$\int \int \text{\$\int \text{\$\approx \text{\$\int \text{\$\approx \text{\$\int \text{\$\to \text{\$\int \text{\$\in

Disappears at

- . 1–2 weeks after abortion
- . 2-8 weeks after labor
- . 8–12 weeks after vesicular mole evacuation

➤ Assessed by

Urine \rightarrow Slide agglutination with latex (detects 500 mIU/ml)

ELISA: more sensitive (90%), (detects <u>50</u> mIU/ml) مراعل المول عرب

 $\underline{\underline{\text{Corum}}} \rightarrow RIA / / \text{ the most sensitive, (detects } \underline{\underline{\text{5}}} \text{ mIU/ml})$

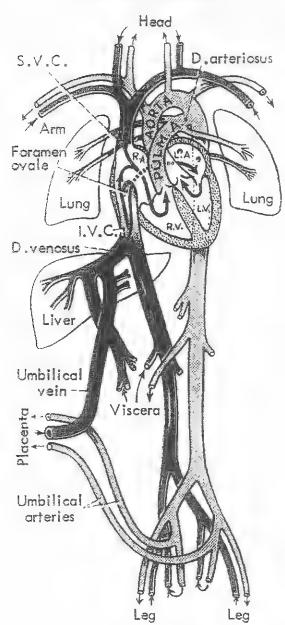
assess β-subunit (as α-subunit is similar to FSH, LH, TSH)

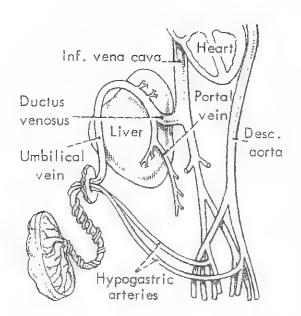
> Value

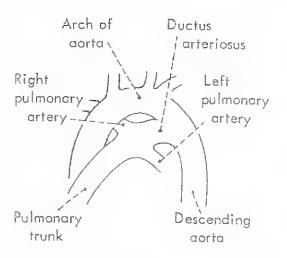
Function	Uses
Maintenance of CL (luteotropic ")	. Diagnosis of pregnancy
Immunological suppressive action	. Diagnosis of pregnancy abnormalitie
Stimulates fetal testosterone sec. *	. Diagnosis and follow up of V. mole

- 2) Human Placental Lactogen (a polypeptide) "
 - > Very similar to GH & prolactin \rightarrow may stimulate growth of breasts
 - > Anti insulin effect on CHO & fat "
 - . Lipolytic \rightarrow metabolism of free fatty acids
 - . Inhibits maternal glucose uptake & gluconeogenesis → spares glucose, fatty acids, amino acids for fetus
- 3) Others human chorionic GnRH, CRH, TRH, ACTH, prolactin, relaxin
 - Placental activin → stimulate GnRH & HCG while inhibin → inhibit them

CIRCULATION







Fetal circulation

O Intrauterine

- Oxygenated blood from the placenta passes to the fetus via the umbilical vein (1) \rightarrow penetrates liver to give it small branches
- Most of the blood is directed via the <u>ductus venosus</u> into the IVC (which carries also the returning non-O₂ blood from LL ⁿ)
- -There is only partial mixing of the 2 streams and most of the oxygenated blood is directed by the <u>crista dividens</u> (at the upper end of the IVC) through the <u>foramen ovale</u> into the left atrium → the left ventricle → aorta → this relatively well O₂ blood supplies → the head & UL:
- The remainder of the blood from the SVC mixes with that of IVC → passes to the right ventricle → very small amount of blood goes to the lungs (high pulmonary vascular resistance [™]). Most blood passes via the <u>ductus arteriosus</u> to the aorta (beyond the vessels supplying the head & upper extremities) → supply viscera & LL
- Little blood actually goes to the LL. Most of it passes into → Rt & lt internal iliac arteries → <u>umbilical arteries</u> (2) *: non O₂ blood

To compensate for the low arterial PO₂ in the fetus:- **

- * Increased fetal cardiac output
- * Increased fetal systemic blood flow rates
- * Increased affinity for O_2 (\uparrow fetal O_2 carrying capacity = Hb-F)

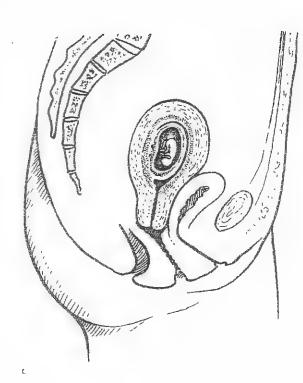
9 At birth

- The umbilical vessels contract in response to \downarrow temp $\rightarrow \downarrow O_2$ tension & $\uparrow CO_2 \rightarrow$ stimulation of respiratory center
- Breathing \rightarrow -ve thoracic pressure \rightarrow sucks more blood from the pulmonary artery into lungs & diverting it from the ductus arteriosus which gradually closes
- The left atrial pressure → closes the foramen ovale

9 Later

- Umbilical vein → <u>ligamentum teres</u> (runs in the free border of the falciform ligament in the adult)
- Umbilical arteries → hypogastric ligaments (lat. umbilical lig) =
- Ductus venosus → ligamentum venosum
- Ductus arteriosus → ligamentum arteriosum

Maternal adaptation



7 weeks PALPABLE UTERINE ENLARGEMENT

At 7 weeks the uterus is the size of a large hen's egg

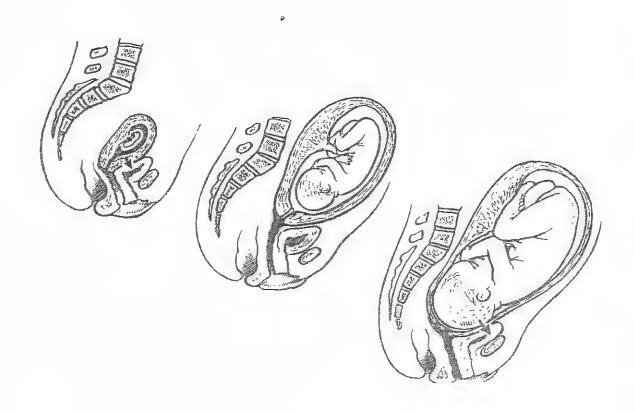


At 10 weeks it is the size of an orange



At 12 weeks it is the size of a grapefruit





<u> Laternal adaptation</u>

The genital tract

The uterus

- \Leftrightarrow Shape $\rightarrow \hat{U}$ from 50 gm (10 ml³) to \rightarrow 1 kg (5000 ml³)
 - \rightarrow changes from pear shape \rightarrow globular \rightarrow pyriform
- - → Then fundal level according to gestational age
 - → Till reaching 35–40 cm at term

\$\frac{1}{2}\to Parition \rightarrow dextrorotated (Lt round ligament becomes nearer to midline)?

→ dextroflexed (d.t. presence of sigmoid colon)

→ soft consistency (d.t. vascularity → 500 ml/min * – & amniotic fluid)

A Myometrium

- o Hypertrophy ✓ & hyperplasia of muscle fibers
- Contractility

 - Later on, cont. are detected abdominally ■ Braxton Hick's
 - They become perceptible & painful near term → false labor pain

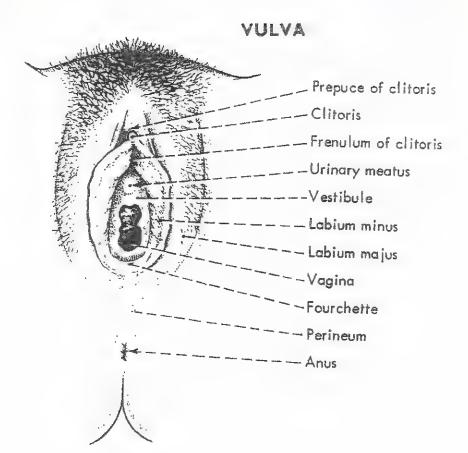
🔯 Formation of lower uterine segment

- Formed from 1sthmus (bet. Anatomical os above & Histological os below)
- It is 4 mm in length and is covered anteriorly by loose peritoneum.
- o During pregnancy & labor → it is stretched to 10 cm
- It differs from the upper segment in

	Upper segment	Lower segment
Peritoneum	Adherent	Loose
Muscle	Thick (3 layers)	Thin (2 layers)
Decidua	Well developed	Less developed
Membranes	Firmly adherent	Loosely adherent
Action	Active in labor	Passive ⁿ
1 1 1 2 2	(contracts & retracts)	(dilates & stretches)

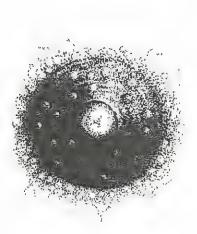
Physiological) retraction ring

It is a groove between the thick UUS & thin LUS below the symphysis pubis. Normally it is not seen or palpable





The breast at 8 weeks



The breast at 16 weeks

2) The ovaries

- No ovulation occurs (suppressed LH & FSH) do +0 1P, E
- The corpus luteum secretes
 - **E&P** → produced mainly from CL till 7 wks, then production is shared with placenta till 10–12 wks, then CL will gradually ↓ in size
 - Relaxin \rightarrow a protein hormone of unknown function. May have a role in ripening of cervix & relaxation of pelvis at labor

• A CL cust may be found in the 1st trimester -> ray be is changed as Ecotopic (< 6 cm disappears spontaneously (functional) frag. by U/s.

• Preg luster—a sold smelling in array the to from Good excess the Greeks in CL-I And

3) The fallopian tubes \Leftrightarrow enlarged, stretched, increased vascularity

4) The vulva

- 1 ded vascularity ⇒ soft & violet (Jacque Meir sign)
- û ed Liability to ⇒ varicose veins & edema

5) The vagina

- û ed vascularity ⇒ soft & violet (Chadwick sign)
- û ed secretions ⇒ acidic (lactobacilli)
- Epithelium is thick (smear → intermediate cells)

6) The cervix

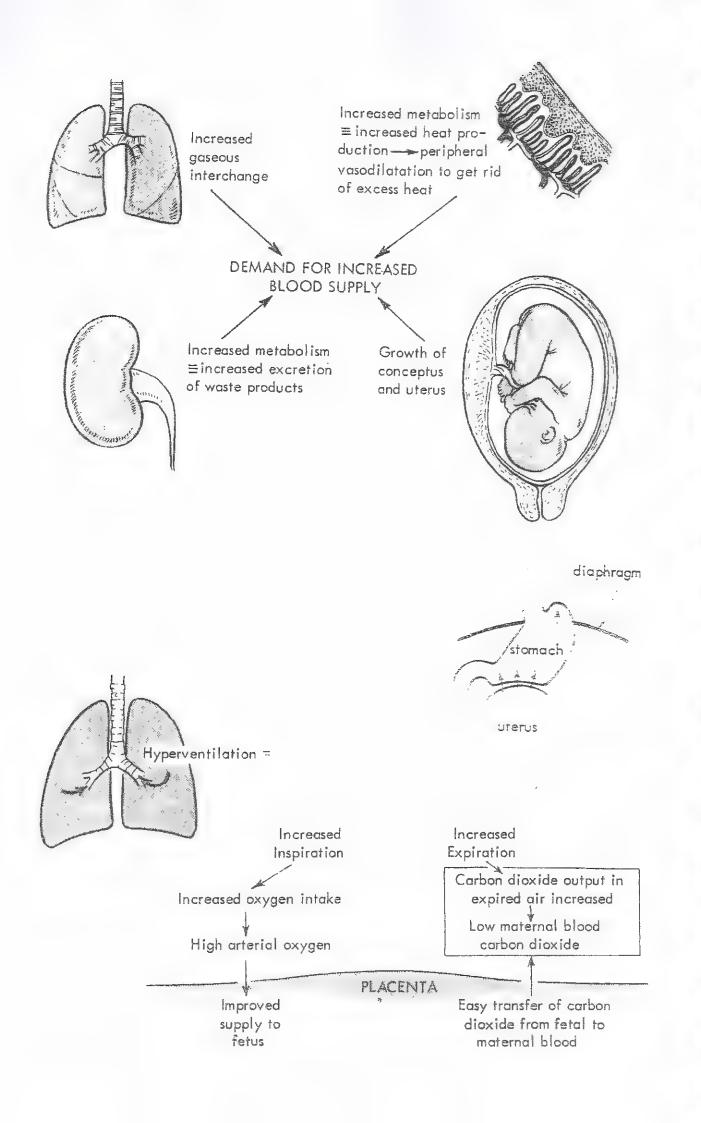
- û ed vascularity ⇒ soft & violet (Goodell's sign)
- $\hat{\Omega}$ ed secretions \Rightarrow mucous plug obstructing cx canal (operculum)
- Epithelium: ectopy (replacement of st.sq.epith of ectocx by columnar)

2 The breast

* The 1st few weeks ⇐> tenderness, tingling (d.t. ↑size, vascularity & sensitivity of nipples & breast)

* The 2nd month

- It ed size & nodularity of breasts she mened.
- û ed pigmentation of 1ry areola & nipple
- Montgomery's tubercles appear (sebaceous glands or pouting lips of the orifices of the peripheral lacteals 10–20)
- * The 3rd month \Leftrightarrow colostrum appears
- * Later on (5th or 6th months)
 - 2^{ry} areola appears (pigmentation around the 1^{ry} areola)
 - The breast shows dilated SC veins & sometimes striae



3 The cardiovascular system

The blood

* Pressure ** \$\mathcal{Q}\$ esp in 2^nd trimester

Progeste one - Reladant home

- Placenta acts as an AV shunt } leading to ♥ in the P.resistance
- & 1 of the peripheral flow - Vasodilator effect of progest. }
- * Volume of Plasma --> increase 40-50% (max at 30-34 weeks)
- * Elements
 - **RBCs** → increase 20–30% ^x

So, there is more \uparrow in plasma volume > RBC volume \rightarrow physiological anemia (haemodilution). Pathological if < 11gm%

- → Haematocrit → decreases
- Leukocytes → û slightly, esp after labor (14–16.000 /ml³)
- Blood coagulation → increased coagualability
 - û^{ed} factors VII–X and fibrinogen + ⊕_{ed} fibrinolytic activity
 - Platelets -> mild decrease (Lemodilution)
- ESR → increases (due to ↑ fibrinogen)
- The heart....changes occur from 1st trimester "
 - ① ed COP (30–50%) → (d.t. ① both SV & HR = 10–15 bpm)
 - The heart is displaced upward & laterally by the diaphragm → shift of apex beat from 5th to 4th intercostal space
 - Due to increased flow rate
 - Heart sounds
 - . Splitting of the 1st sound
 - . Appearance of the 3rd sound by pe dynamic Circulation
 - Heart murmurs
 - . Soft systolic murmur may be present (90%)
 - . If diastolic murmur occur we must exclude pathology
- Veins There is increased liability to varicose veins due to more in 4 22 due to fresture - Progesterone (relaxant effect on vessels)
 - Pressure of the gravid uterus
 - Increase in blood volume

f 4 The respiratory system

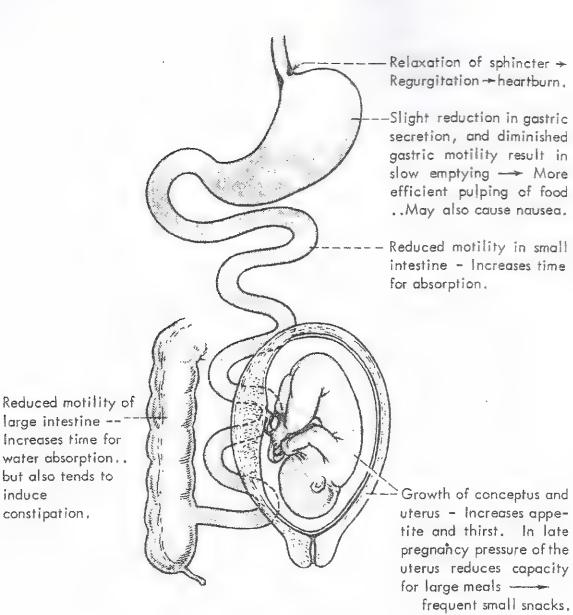
of effection - 1RC - shape vertilation o û ed dyspnea in late pregnancy → pressure & hormonal effect

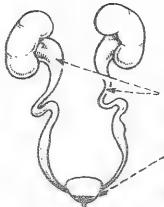
û ed tidal volume → reserve volume decreases

o û ed minute respiratory volume } progesterone effect 〕

o î ed minute oxygen uptake } resp. alkalosis = the to type neutilation -> Ca wash.

No change . What about the " RR.Q)





induce

constipation.

The ureters are said to dilate greatly and again urine may stagnate.

Relaxation of bladder may result in incomplete emptying and collection of residual urine.

Both of these changes tend to favour the onset of urinary infection.

Spe gastrointestinal trace

1) The mouth

- Changes in appetite

 longing (pica)

 desire to certain food

degree that affect great and tom (Known Kliketon bodie: in wine).

2) Esophagus

Relaxation of cardiac sphincter
 Delayed gastric emptying
 leads to pyrosis
 heart-burn (P. effect)

3) Stomach & intestine

- Delayed emptying, decreased motility
- ↓ acidity of stomach (hypochlorohydria) → regurgitation of alkaline chyle
- 4) Liability to constipation & piles \Rightarrow d.t. effect of Pr & pressure of uterus

5) The liver & gall bladder

- Changes in some liver enzymes esp alkaline phosphatase
- Tendency to cholestasis (1Pr) -18ile salts -> italing.
- û ed globulin + Ded albumin Gallinding globalings +1 IGs.

3 The urinary tract

➤ The kidney

- o Increased size (by 1-1.5 cm)
- o Increased renal blood flow → increased GFR (50%) $^{\text{m}}$
 - \bullet ↑ creat. clearance \rightarrow ↓ serum creatinine (0.5 mg%), uric (3 mg%), BUN (8.5 mg%)

The urefers

- o Enlarged, dilated & tortuous (d.t. progesterone & pressure at pelvic brim)
- More on right side (d.t. dextroflexion of uterus)
- o Increased liability to pyelonephritis

> The bladder

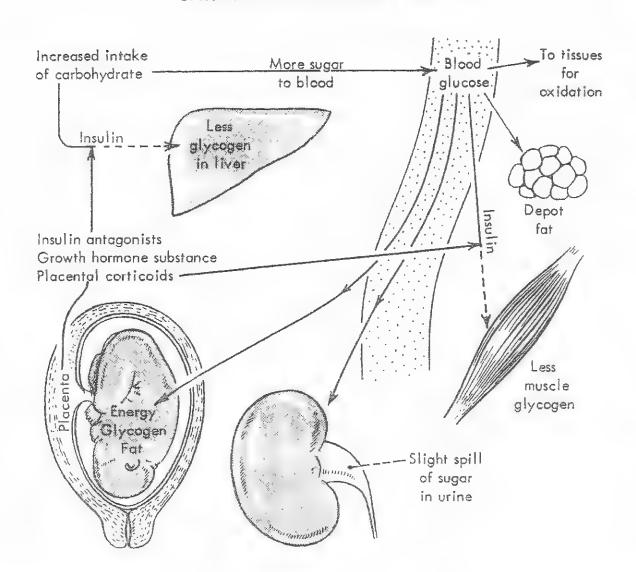
- o Hyperemia
- ↑ frequency of micturition (1st trimester) & (last month d.t. engagement)
- Displacement upwards (SUI may be normal in late in pregnancy)

7 The nerve & muscle

- Mood changes (elevated or depressed) (sleepy or insomnia)
- Relaxation of the pelvic joints, sometimes arthropathies (Relaxin h).
- Lumbar Lordosis → to compensate for the enlarged uterus



CARBOHYDRATE METABOLISM



A all Lermones.

The all binding globulins.

There level of all her monosare not changes.

3 The endocrine glands

> The pituitary

Increased size & vascularity (esp anterior lobe: 2-3 folds)
↑ prolactin – ↓ FSH & LH, GH [other hormones are unaffected]

> The thyroid

Slight enlargement (↑ TSH & chorionic thyrotropin)

↑ serum thyroxine [¤] (due to ↑ TBG, free hormones are <u>unchanged</u>)

1 BMR 25% (due to pregnancy & not hyperthyroidism)

> The parathyroid

Slight enlargement → ↑ PARATHORMONE to ↑ serum Ca→Provide Ca for fetus. but CALCITONIN also ↑ .. no change in ionized Ca level

> The suprarenal gland

Little morphological changes

† aldosterone & renin (due to † CBG, free serum cortisol are unchanged)

9 The skin and appendages

1. Pigmentation.....d.t. 1 placental & adrenal steroids, also E. may have MSH like activity

c Esp in nipple, areola, axilla, vulva

- <u>Linea nigra</u> (dark brown line between umbilicus & symphysis)
- o Chloasma gravidarum (butterfly pigmentation on face)
- 2. Striae gravidarum.....due to û corticosteroids or by mechanical stretching
 - ⊃ Pink lines due to rupture of elastic fibers or SC vessels of skin of abdomen (common), breast, thighs, buttocks. Later on after delivery → striae albicans
- 3. Divarication of recti
- 4. Hyperemia → vascularity of skin & m.m. (nasal congestion) + Comer epictostis.
- 5. Sometimes → falling of hair, palmar erythema and spider naevi

10 Merabolic changes

- ▶ Body weight ⇒ increases 12.5 kg on average /9-12.5 kg)
- ▶ Protein metabolism ⇒ +ve nitrogen retention (1 kg increase during pregnancy)
- ► CHO metabolism pregnancy is potentially diabetogenic
- ightharpoonup Lipid metabolism ightharpoonup increased blood lipids & cholesterol ightharpoonup central fat deposition
- ➤ Minerals ⇒ serum iron (Fe stores may be depleted if no Fe supplementation is given)

 But ↑ transferrin (total iron binding capacity)

 The only modern of stores required in

Minor Disturbances in pregnancy

O Gastrointestinal

- > Morning sickness....
- ▶ **Gingivitis:** hyperemic gums that may bleed with the use of a tooth brush
- > Ptyalism: excessive salivation
- ▶ Heartburn: Treated by antacids, more frequent meals, avoidance of spices
- ▶ Indigestion: hypochlorhydria (regurgitation of alkali chyle into stomach)
- ➤ Constipation: û fluid intake ± eating whole meal bread [& not white bread]
- ▶ **Hemorrhoids:** usually regress after delivery [but not completely]

9 Urinary

- Frequency of micturition: d.t. pressure from the gravid uterus
- ▶ Incontinence: d.t. loss of the post urethra-vesical (PUV) angle

Musculo-skeletal

- Backache:
 - Common in the last trimester
 - Treatment: . Avoid wearing high-heeled shoes
 - . Exercises to strengthen the back muscles
- ▶ Leg cramps:
 - Electrolyte disturbance
 - Engorgement of lower limb veins
- > Round ligament pain:
 - Sharp groin pains d.t. spasm of the ligament associated with sudden movements (esp the right side ---d.t. dextroposition)

O Skin changes

- Striae gravidarum
- > Sweating & feeling the heat: (d.t. û peripheral circulation &VD)
- ➤ Vaginal discharge = leucorrhea: (d.t. û estrogen)

1 Nervous system

- ▶ Insomnia d.t. the large uterus, leg cramps & backache
- **▶ Carpal tunnel syndrome:** d.t. edema → disappears 2 wks after delivery
- ▶ Placidity (calmness) & drowsiness: d.t. û progesterone

© Cardiovascular symptoms

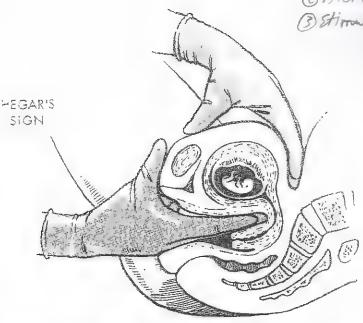
- Varicose veins: treated by :
 - Patients should sit with their feet elevated whenever possible
 - Nylon elastic stockings should be put in on the morning before getting out of bed......to be removed on sleeping
- Headaches, palpitations & fainting
- Physiological edema (below knee)

Benitti de Cond.: (2 y pmp).

O enhans vi-blod Plor. (2 y pmp).

O Aid aminiotte Pluid secretion

(3) Stimulete Petal novement.



KEY POINTS

- 1. A urine pregnancy test will often be positive at the time of the missed menstrual cycle.
- 2. Physiologic changes during pregnancy, mediated by the placental hormones, affect every organ system.
- 3. Cardiovascular changes include a decrease in systemic vascular resistance and blood pressure and a 50% rise in total blood volume.
- Elevation in serum progesterone levels is responsible for smooth muscle relaxation in the vascular system, GI tract, and genitourinary system, leading to many of the concomitant physiologic changes.

The first n	ine weeks		
Days	Weeks:	Clinical features	Scan features
0	0	Menses	
7	1		
14	2	Conception	
21	3		
28	4	Pregnancy test positive [menses due]	Empty uterus
	5		Gestational sac (hCG >2000 IU)
	6	Nausea Breast tenderness	Yolk sac, Fetal heartbeat on transvaginal scan Fetal pole 4 mm
	7		Fetal pole 10 mm
	8		Fetal heartbeat on transabdominal scan Fetal pole 14 mm
	9		Fetal pole 22 mm

Diagrosis of pregnancy

In the 1st trimester -

Sumptoms

- Amenorrhea (Not a sure sign)
 - may have amenorrhea due to other causes
 - may have bleeding in early pregnancy
- Breast symptoms as heaviness, pain, enlargement, colostrum
- Morning sickness....Appetite changes...Frequency of micturition
- Some ladies may experience fatigability & sleepiness, while others may have irritability & insomnia

- · Breast signs & Browning of areda.
- Genital signs

Vulva (soft & violet) www..... Jaque-Mier sign Vasculoria Vagina (soft, warm & violet)..... Chadwick sign

Cervix (enlarged, soft & violet)... Goodell sign

- Uterus → * Enlarged & soft

* Change in shape

*Condractliguterus. Palmer sign - Binamal ex.

Hegar sign (d.t. softening of isthmus) Two fingers between the ant. vaginal fornix & abdomen behind the uterus can be approximated (between 6-12 wk)

. < 6 wks \rightarrow uterus is not soft enough

 $. > 12 \rightarrow$ the baby occupies the whole uterine cavity

> Investigations

- Pregnancy tests
 - * Immunological [biological are obsolete]
 - <u>Urine tests</u> (conventional pregnancy tests) ⇒ Latex & ELISA
 - Serum test: β-subunit (most sensitive)

 RIA
- Ultrasonography

 - Transabdominal U/S ≈ 7 wks
 - Detection of cardiac activity \approx 8 wks
- ♦ Biochemical pregnancy means detection of +ve β-HCG before missed period
- ♦ The window gap

The gap (2 weeks) between.....Biochemical pregnancy (3rd wk) & TVUS visualization of pregnancy (5th wk)





A Pinard stethoscope.

History

Pinard's stethoscope was invented in France in 1816 by René-Théophile-Hyacinthe Laennec (1781–1826). It consisted of a wooden tube and was monaural. His device was similar to the common ear trumpet, a historical form of hearing aid; indeed, his invention was almost indistinguishable in structure and function from the trumpet, which was commonly called a 'microphone'.





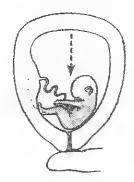
Using the Pinard stethoscope,



Tap gently upwards and hold finger against cervix



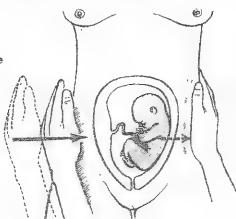
The fetus is displaced upwards



The fetus sinks and a gentle tap is felt on the finger.

24 EXTERNAL BALLOTTEMENT

One hand taps the abdomen and sends the fetus across the uterine cavity.



The other hand lying on the uterus perceives the impulse.

In the 2nd trimester

▶ Symptoms

- Amenorrhea.....Breast symptoms increase
- Quickening (1st perception of fetal movement)
 - In PG \rightarrow 18 20 weeks
 - In MG \rightarrow 16 18 weeks
- Progressive abdominal enlargement

Signs

- Breast signs
- Uterine signs
 - * Braxton Hick's contractions
 - * Uterine soufflé may be heard → soft blowing murmur Synchronous to the maternal pulse (due to increased blood flow through the dilated uterine arteries)
- Fetal signs

Ballottement (due to movement of fetus within amniotic fluid) - Internal ballottement → between 16 – 28 weeks

- External ballottement \rightarrow > 24 weeks

* Palpation of fetal parts (after 24 weeks)

* Inspection or palpation of fetal movement

*Auscultation of.....fetal heart sounds by Pinard stethoscope (18 wk *)Umbilical soufflé (funic soufflé) -> Soft whistling sound -> Synchronous with the fetal heart sounds. It is due to flow of blood in the umbilical vessels and is heard sometimes when a loop of cord is in a close proximity to the anterior uterine wall

▶ Investigations (as in 1st trimester) → less needed as diagnosis is usually easy

Sure signs of pregnancy

- INSPECTION OF FETAL MOVEMENT
- PALPATION OF FETAL MOVEMENT / PARTS
- AUSCULTATION OF FETAL HEART SOUNDS OR UMBILICAL SOUFFLÉ
- Ultrasonography or X-ray to Visualize the Fetus

DD of pregnancy: causes of:

- Amenorrhea
- Symmetrical enlarged uterus
- Don't forget pseudocyesis

Anternatal Care

Antenatal care

☆ Objectives

- To try to get a healthy mother & newborn
- Estimation of gestational age & expected delivery date
- Early detection & treatment of any diseases during pregnancy
- Early detection of congenital fetal malformations

☆ Consists of

- History taking
- Physical examination
- Investigations → Routine & screening tests
 Other investigations according to findings
- Plan for a schedule for return visits
- Instruction & advice
- Reassurance
- Plan for delivery

High risk pregnancy

Aim of ANC is to detect or suspect any conditions that may lead to maternal or fetal hazards i.e. to detect <u>high-risk pregnancy</u> To

- . Pregnancy associated with increased risk
- . Whether (maternal or fetal)
- . Due to certain risk factors:

O Socioeconomic

- Socioeconomic status
- Parental occupation
- Psychological e.g. excess anxiety → preterm labor

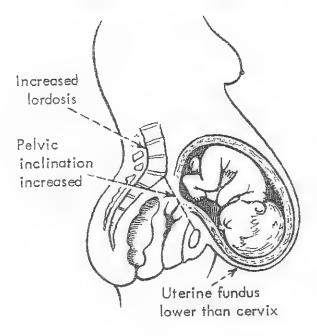
Demographic factors

- Maternal age (optimal age is between 20–30 yrs)
- Maternal education

1 Medical factors (disease)

. PET	. Anemia	.DVT
.DM	. Renal disorders	.Thyroid disorders
. Heart disease	. Hepatic	. Respiratory
. Hyperemesis	disorders	disorders
gravidarum		

GRAND MULTIPARITY



1 History

> Personal history

- Name triple name
- Age.....lowest rate of MMR & PNMR is 20–26 yrs.....

Adolescent pregnancy	Pregn. in old age (>35 yrs)	
.Nutritional deficiency (immaturity)	.Nutritional def (consumption)	
.Hypertensive disorders .HTN + DM		
.Dystocia (small pelvis??)	.Dystocia (osteomalacic pelvis??)	
.Social & economic .Chromosomal Down syndron		
+ + Abortion, IUGR, PTL		

- · Marital status
- Parity higher MMR & PNMR in
 - 1. Grandmultipara (≥ 5 deliveries) \rightarrow liable to $\Phi\Phi\Phi$

Pregnancy	Labor
.Abortion, PTL, anemia	.Uterine atony (more fibrous tissue)
.Malpresentation (lax abd. wall)	.Obstructed labor → rupture uterus
.Placenta previa (accreta)	.РРНде
.Chronic hypertension, DM	

2. Elderly PG (\geq 35 yrs) \rightarrow liable to $\Phi\Phi\Phi$

Pregnancy	Labor		
Abortion, PTL	.Prolonged labor (ப் ^{ed} maternal		
.Chromosomal anomalies (Down)	anxiety & abnormal ut. action)		
.Hyperemesis gravidarum	.Rigid perineum → episiotomy		
.PET \rightarrow P.abruption, DM	.Higher rate of CS		

- Address => social conditions & environment
- Occupation \Leftrightarrow certain occupations have certain risk e.g.
 - Medical personnel \rightarrow infectious diseases, anesthetic gases
 - Others → radiation (factories)

Special habits

- * Smoking ⁿ → abortion, IUFD, IUGR, perinatal death, APHge, oligoamnios
- * Alcohol $^{\pi}$ \rightarrow abortion, IUFD, IUGR, perinatal death, CFMF, mental handicap
- * Narcotics → fetal depression & addiction
- * Pets \rightarrow risk of toxoplasmosis

> Menstrual history

- LMP \rightarrow important for dating of pregnancy (EGA) & calculation of EDD
- Must know if it is average, regular, if sure of dates or not, if pregnant on period of amenorrhea, or after COC

> Obstetric history

Number	. Prolonged period of 2 ^{ry} infertility	
Year of birth	. Rapid succession → liability to malnutrition	
Place of birth	Previous uncomplicated home deliveries → reassuring	
Antepartum period	 Repeated hypertension → expect recurrence Previous DM → screen for DM Previous APHge or PROM → may recur 	
Duration of preg.	Previous PTL → suspect maternal or uterine disease	
Onset of delivery	Spontaneousinduced	
Mode of delivery	Easy vaginal delivery → expect another - If previous complicated → plan for possible CS - Forceps or ventouse → suspect CPD Cesarean section → why?	
Postpartum comp.	PPHge	
Baby	. Alive, incubated, malformed, dead . Male / female . Weight . Breast / bottle fed	
Puerperium	Puerperal sepsis, DVT	

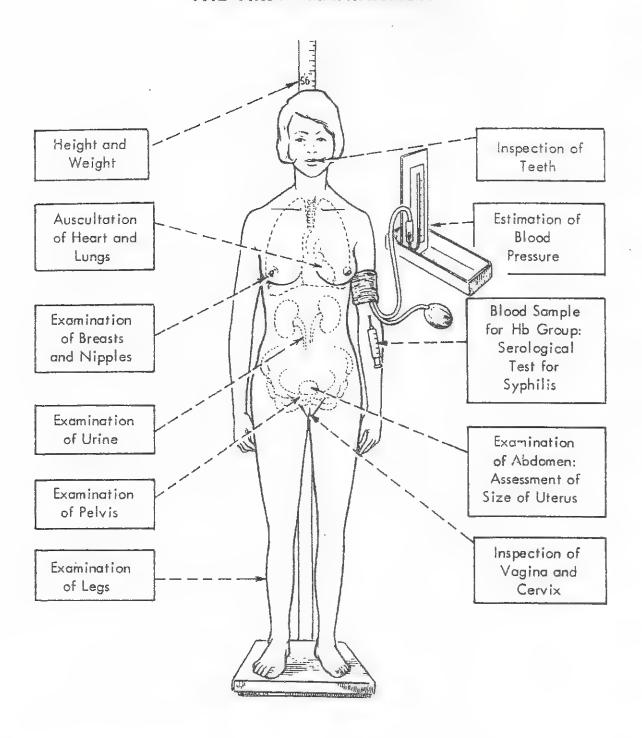
> Past history

- Medical → DM: screen.....Hypertension: investigate.....DVT: prophylaxis
- Surgical→ Previous operations
- Drugs → May affect pregnancy or fetus e.g. oral anticoag.
- Previous blood transfusion
- Presence of allergy to drugs

> Family history

- DM → screen
- Hypertension → investigate
- CFMF → screen
- Twins → suspect

THE FIRST EXAMINATION



O listory

Nikolai Sergeivich Korotkov (1874–1920) was a Russian physician who devised a method of measuring diastolic blood pressure by applying the stethoscope to the brachial artery during the deflation of a sphygmomanometer cuff.

Examination

مانور الري General

- Decubitus: dyspnea
- Height: if less than 150 cm → be aware of CPD → Captalo-Pelvic disproportion
- Weight: if obese beware of D.M., hypertension, macrosomia & dystocia

3%	No	rmal	W	eig	ht	gain
	=	12.5		13	kg	3

- * Underweight women $= BMI < 20 \text{ kg/m}^2$
- * Overweight women $= BMI > 26 \text{ kg/m}^2$

		33
Fetus	3500	
Maternal fat	3500	
-Blood	1500	
-Extravascular fluid	1500	
Uterus	1000	
Amniotic fluid	1000 maxis	men anjust
- Placenta	1000 - mayi, 500 Bluje	Jed-ete (/
- Breasts	300	Calcificat or
Total	12.5 – 13 kg	

☆ 3 vital data

- **B.P....** for hypertensive > 140/90 how??
- Pulse......abnormal pulse, esp. in heart disease
- Temperature...esp. in infection or PROM

☆ 3 colors

- Jaundice ... pregnancy associated or induced ATN, Hyperenisis gaurden.

 Cyanosis > Fiser menger 8.

 Acute fatty Uner

 Palloranemia, bleeding with pregnance.

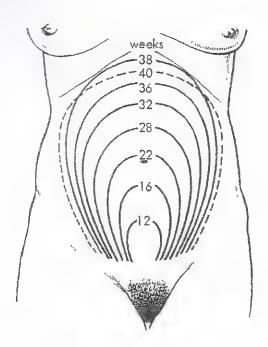
- Thyroid
- **Vessels**....engorged normally (↑ blood volume)
- LNs

• Chestchest infection, PVC

......what are sure signs of pregnancy?

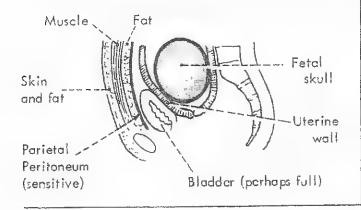
Breastnormal changes in pregnancy, galactorrhea

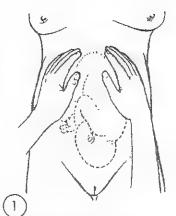
- Gait (look for limping → CPD)
- Back
- Lower limb for varicosities, DVT and edema.



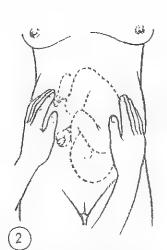
This examination must be made systematically.

Remember that the following tissue layers may interpose between your fingers and the fetal head.

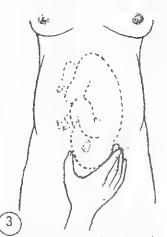




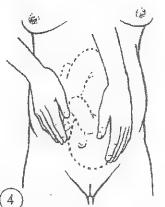
The fundus is palpated and the breech identified.



The hands palpate the contours of the uterus, identifying the back and the limbs.



The head should be palpated, and it should be noted whether it is mobile or fixed in the pelvic brim.



The examiner faces the patient's feet and gently pushes two fingers into the pelvis. This is the best method of palpating the fetal head and determining whether it is fixed or mobile.

> Abdominal

1. Inspection

Size → huge (twins or polyhydramnios)	Movement with respiration
Shape \rightarrow if pendulous in PG \rightarrow cont. pelvis	Pigmentation → linea nigra
Striae, veins, scars	Hernial orifices
Supra-pubic hair → feminine / masculine	Umbilicus

2. Palpation (4 Leopold's maneuver)

☆ Fundal level by hand or in cm above S.pubis

☆ Fundal grip	WEEKS	LEVEL
- Cephalic or breech	12	Symphysis pubis
- Empty transverse lie	16	na taon kadamanana di Senamo tamon Monamanana Makakaman kalajanapap (Pipinasaya asiin at alajangan asap)
☆ Umbilical grip	20	
- For lie	24 (20-22)	Umbilicus
- For back & limbs	28	
- For amount of liquor	32	
- Expected fetal weight	36	Xiphi-sternum

- For any local uterine swelling
- ☆ 1[±] & 2nd Pelvic grip (Pawlick's grip)
 - To determine presenting part (head, breech, empty in tr. lie)
 - To determine head engagement
 - To determine degree of flexion of the head e.g. extended in face

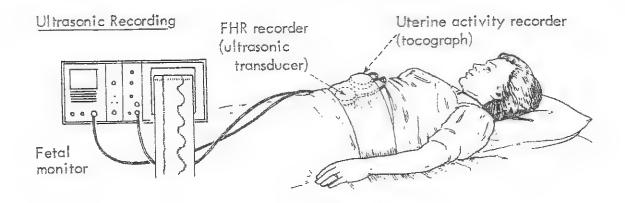
3. Auscultation

		-1
ATRICIT CONTRACTOR	ş	
	z z	,
The state of the s	× /	
Mar in	W.	

an be used for	Different sites for FHS
n of pregnancy	. Normal (OA) \rightarrow between umbilicus & ASIS
e / distress	. O.P. $ ightarrow$ at ASIS
Arnoux sign	. Face \rightarrow MA at $<$ umbilicus, MP at flanks
s in labor	. Breech \rightarrow complete $>$ umb, frank $<$ umb
& presentation	. Transverse lie at one side
	s in labor

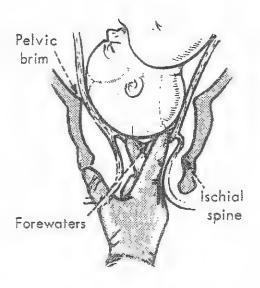
> Local

Done	In early pregnancy for	In late pregnancy for
only	- Diagnosis (Hegar's sign, Ballottement)	- To diagnosis labor
at	- In some complications e.g. ectopic preg	- To assess pelvis for CPD
ai	- Any associated pathology e.g. prolapse	- Any associated Pathology
	- To take cervico-vaginal smear	



causes of non-engagement of head in P.G. D.

Fetal	Maternal
- Large head, Hydrocephalus	- Contracted pelvis
- Malposition or malpresentation	- Tumor in pelvis
- Multiple pregnancy	- Placenta previa
- Short cord	- Full bladder or rectum
- Polyhydramnios	- No cause may be found



8 Investigations

> Routine:

- o Blood for: "
 - Blood group & hemoglobin %
 - Rh (If Rh -ve see husband......if multipara determine if sensitized)
 - Blood sugar at 24 28 weeks
 - Hepatitis B surface antigen
 - Serology for syphilis
 - Antibody to rubella
- o Urine for: Glucose.....Protein.....Bacteriuria (not Cas")

Specific:

- o According to history and examination
- o Ideally \rightarrow U/S \pm FWB tests in high risk pregnancy

4 Return visits

- Every 4 weeks till 28 wks
- Every 2 weeks till 36 wks
- Every 1 week till delivery
- If any abnormality present → more frequent visits
- At each return visit
 - ☆ History: Ask about any symptoms esp. fetal kicks >

Dangerous symptoms *

....In early pregnancy

- Bleeding, Pain, discharge (watery or infected)
- Fever, Dysuria
- Persistent vomiting

....In late pregnancy (as above +)

- Symptoms of PET (blurring of vision, epigastric pain)
- symptoms of DM (polyuria, polydypsia, pruritis)
- Change in intensity or decreased fetal kicks

☆ Examination

- General ⇒ weight / blood pressure / edema
- Abd fundal height, liquor amount, presentation, position, FHS
- PV = late or in presence of abnormality (not essential).

1 Investigations

- Routine . Urine in 3rd trimester for glucose, protein
 - . Hb% is repeated at 34 weeks
- - . α -fetoprotein (16 wk) for suspected anomalies
 - . vaginal swab for chlamydia or bacterial vaginosis

Dietary advice in pregnancy

Food to avoid

Risk

Soft cheese

Listeria: fetal infection can lead to miscarriage or

Unpasteurized milk and

cheese

stillbirth

disability

Uncooked fish, e.g. sushi, smoked fish

Unwashed salad/fruit/

vegetables

Toxoplasma: fetal infection can lead to miscarriage, stillbirth, or long-term

Raw and rare meat

Unpasteurized milk

Shellfish

Uncooked eggs

Can cause food poisoning, which can precipitate premature labor

Dietary advice in pregnancy.

Foods that carry potential infection risks in pregnancy

Soft cheeses

Unpasteurized milk and its products may contain listeria. Those made from

pasteurized milk are safe

Raw eggs

Must be avoided as there is a risk of salmonella (including puddings)

Meat or pâté

Undercooked meat may transmit toxoplasma or rarely listeria

Fruit

This should always be washed before eating as it may be contaminated with salmonella, toxoplasma or one of several

intestinal parasites

6 Instructions & advice

> Nutrition

- Caloric requirement → 2200 2500 K.cal / day
- Daily increase of 300 K.cal (esp. in late pregnancy)
- Meals should be well balanced & discourage overeating
- If diet is adequate → no need for supplementation (except...)

1. Proteins

Requirement 1.5 g/Kg/d \rightarrow addition of 1 kg protein to body weight Best if from animal sources (esp. milk)

- 2. Carbohydrates → to complete the caloric requirement
- 3. Fats \rightarrow to complete caloric requirement
- 4. Vitamins

A → 5.000 IU /d	Ascorbic acid (vit C) → 100 mg /d
$D \rightarrow 400 IU /d$	Vit K → PPHge & fetal Hge
$B_1 \rightarrow 1 \ mg \ /d$	Folic acid $\rightarrow 0.8 - 1$ mg /d $^{\text{x}}$
$B_2 \rightarrow 1.5 \text{ mg/d}$	Nicotinic acid \rightarrow 1.5 mg /d

5. Minerals

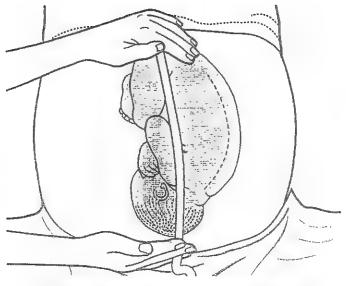
- Calcium: 1 g /d (2 cups of milk)....supplementation is not essential "
- Iron: 30–60 mg /d......the only supplementation required I $^{\alpha}$ \pm folic acid
- <u>Salt</u>: no need for either supplementation or restriction (except in HTN)

Rest

- o At least 8 hrs at night & 1-2 hrs in the afternoon, better on her left side
- o Helps to increase placental flow
- ► Exercise → allowed in moderation esp walking in fresh air & swimming
- ► Employment → allowed until delivery unless physically demanding

> Travel

- Allowed, but if > 6 hrs... walking / 2 hrs to avoid DVT
- o Better avoided in last month
- **Clothing** → loose unrestrictive, better no high heel
- **Bathing** → allowed & encouraged esp tub baths (less liable to accidents)
- **Douching** → high vaginal douching is condemned "...increases infection
- **Sexual activity** → allowed unless there is:
 - o Hemorrhage, risk of abortion or PTL, infections, ROM
- **Coffee & tea** → no harm (but excess \rightarrow irritability & \checkmark Fe absorption)
- **> Smoking** → discouraged
- ➤ Alcohol → discouraged
- > Care of teeth → as usual
- ➤ Medications → should consult the obstetrician before receiving drugs.



Measurement of symphysiofundal height.

Obstetric diagnosis

- ▶ Name, Age, Para + , Pregnant at wks
- ▶ Presentation (cephalic, breech), not in labor
- Complication (obstetric..... medical)

Calculation of EDD

> History

- 1. Menstrual delivery interval: 'calculated from the 1st day of LMP'
 - 280 days.....or....40 weeks
 - 10 lunar months...or....9 calendar m + 7 days
- 2. Naegel's rule " 'but on 3 conditions'
 - 1^{st} day of LMP + 7 days + 9 months or
 - 1^{st} day of LMP + 7 days 3 months
- 3. Fertilization delivery interval I know the day of fertilization
 - Coital delivery time e.g. in IVF or rape (timed event)
 - The duration is 266 d or 38 wk or 9 m 7 d
- 4. Quickening > PG (18–20 wk).....MG (16–18 wk)

> Examination

1. Fundal level $\Phi \Phi$

Causes of FL > amenorrhea Causes of FL < amenorrhea 1- Miscalculation 1- Miscalculation 2- Pregnant on period of hge 2- Pregnant on period of amen. 3- Multiple pregnancy 3- Missed abortion 4- Macrosomia 4- IUGR 5- Oligohydramnios 5- Polyhydramnios 6- IUFD 6- Concealed accidental hge 7- Tumors: fibroids, V.mole 7- Transverse lie

- 2. Symphyseo-fundal height.....'McDonald rule'
 - Wks of pregnancy = height of fundus (in cm) $\times 8/7$
- 3. Gravidogram
 - Progressive ↑ in FL above SP (1cm/wk after 20 wk)
- 4. Abdominal girth
 - Circumference around the umbilious in inches = wks of preg
- 5. Pinard stethoscope detects FHS at 18-20 wks

Investigation

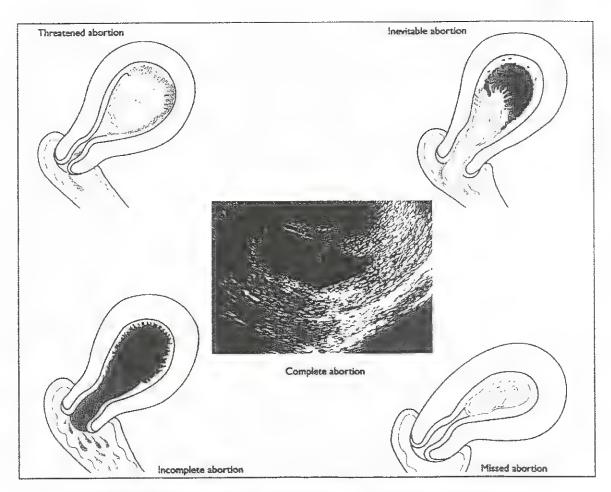
- 1. Ultrasound.....esp the 1st trimester (the more accurate)
- 2. **Doppler**......10 wks

Chapter 6)

Bleeding in early
Pregnancy

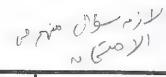
Abortion Ectopic

Vesicular mole



The types of abortion which may be seen. In complete abortion, the sac contains a small amount of debris.

Obstetric hemorrhoge



Early	y pregnancy Antepartum hge		Post P. hge	
Mainly	Others	Placental	Extra-placental	- Atonic
- Abortion	- Local gynecolog.	- Placenta	. Fetal: vasa previa	- Traumatic
 Ectopic 	conditions	previa	. Rupture uterus	- Retained pl.
 Vesicular 	- Hartman's sign	- Abruptio	. Excessive show	- DIC
mole	- Decidual hge	placenta	. Marginal sinus bl	- Acute inversion

Definition → termination / interruption of pregnancy <u>before</u> period of fetal viability (gestational age at which fetus is

capable of extra-uterine existence) i.e. (20 weeks = 500 gm) / in developed countries & depends or (28 weeks = 1.000 gm) in developing countries the level of the NICI facilities.

Types

Spontaneous	Induced
Threatenedmissed ←↓ 3 septicinevitable	● medical indication→ Therapeutic
 ⇒ . ⑤ complete . ⑥ incomplete . ② cervical ⑧ If recurrent > 3 times ⇒ Habitual 	non-medical indication ∴ Elective (voluntary) Criminal



Incidence 👄

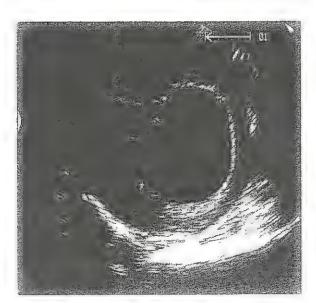
- 15-20 % " mostly in the 1st trimester esp. in the 3rd month 8-12 week (due to some 4 in 'P' from C.L., while placenta still not fully developed yet; the window gap)
- True incidence may be much more (50–80%) due to: - Subclinical abortion (very early < patient recognition)
 - Notification is not done in all cases (esp illegal)
- Incidence increases with ^a
 - *Increase* in maternal & paternal age
 - Previous abortions or stillbirth or CFMF



intrautavine gast-wonal stak thereming a 6 millional with a your factor.



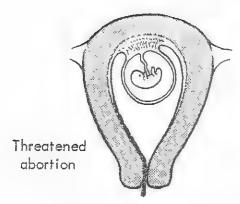
An intracterine 32 mm (see, only turks in 1979). Weeks gestation 1000



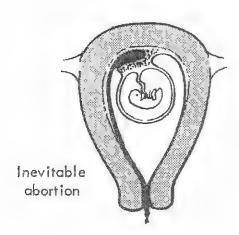
Mu Brown Chrotister and a second second second

og Cetal mal Formation Etiology ΦΦΦ A] Fetal (CFMF)malformed fetus The commonest cause (50–60%) ⁿ of 1st trimesteric abortion May be $^{\pi} \rightarrow trisomy$ (50%), polyploidy (25%), monosomy x (15%) Blighted own (anembryonic sac) is a type of CFMF in which severe and the fetus. fetal tissue is replaced by homogenous structureless sac 1. Maternal disease as hypertension or chr. renal disease 2. Endocrinal (25%) - \downarrow progesterone \rightarrow C. Luteum or placental insufficiency - ↑ androgens -> PCO Panares - Other hormonal dist. → DM, hypo- or hyper-thyroidism, A da in early Pregnancy 3. Infections (STORCH) ... Any organism causing high fever e.g. typhoid fever... due to 1 PGs - Bacteria → Syphilis, mycoplasma, chlamydia, listeria Viruses → acute viral infection, rubella, CMV - Protozoa → toxoplasma?!, malaria 4. Immunological Antimolean Ho - Autoimmune - SLE, APS - Anti Phospholipid & Alloimmune → Rh isoimmunization 5. Drugs (cytotoxics) or Chemicals (heavy metals) or Radiation (>5 rad) 6. Frauma → direct surgical (CL removal by mistake in appendectomy) . Coso Sue of Le Jim on Stesh fetus 1. Cewix Patulous internal os 2. Uterus - Congenital malformation: septum, hypoplasia - Small cavity: submucous fibroid or Asherman's \$ - Limited distension: fixed RVF Overdistension: acute polyhdramnios Pathogenesis PG, > 1^{s'} trimester □ Triad of • amenorrhea من في bleeding من الترتيب مهم وبر و Triad of • amenorrhea Usually bleeding occurs into the decidua basalis (choriodecidual hge) \rightarrow uterine irritation \rightarrow colic \rightarrow expulsion of the pregnancy sac (either intact or as fragmented parts) > 2nd trimesteric abortion \square Usually......amenorrhea \rightarrow bleeding \rightarrow pain, or \square Sometimes.....amenorrhea \rightarrow ROM \rightarrow pain (contractions) Anniotic fluid 15 full of & Senere Pain.

PGs -> stimulates utzus -> severe Cont.



Bleeding is slight, not retroplacental, and cervix is closed. Pregnancy is likely to continue.



CI befor 18 weeks as there is no B-receptors HU 18 weeks.

- Dilatation / effacement of the cervix progressively

Turned into septic, missed

Rupture of the membrane, partial protrusion of products of conception

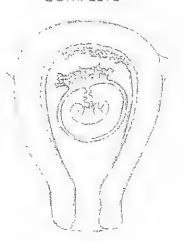


nations leading to local necrosis and inflammation.

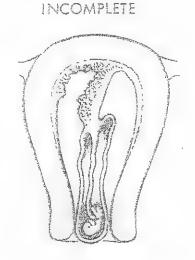


The cyum, partly or wholly detached, acts as a foreign body and initiates uterine contractions. The cervix pegins to dilate.





or



Expulsion complete. The deciduals and during the next few days in the point! How.

2) Inevitable abortion

معنو « لوقت Complete separation of the fertilized ovum with progressive cervical dilatation & fetal expulsion

> Symptoms

- Amenorrhea + symptoms of early pregnancy
- Bleeding → moderate to severe
- Pain → marked LOWER abdominal COLICKY pain (uterine contractions)
 with BACKACHE (cervical diatation) = sacral pain * duto fraction en

▶ Signs

- General → Pallor / shock (according to amount of bleeding)
- Abdominal → uterus corresponds to period of amenorrhea
- Local → OPENED CX (products of conception may be protruding)

> Treatment

1. Resuscitation if bleeding is excessive

2. Evacuation 1st trimesteric → evacuation by suction or curettage

3. Followed by . Echolics -> helps complete evacuation of remnants -> (dicidual)

(republic Antibiotics -> reduces possibility of postabortive infection.

4. Anti-Dif Rh ve

3) Complete abortion

- ➤ <u>Definition</u>

 all products of conception have been expelled
- ➤ Symptoms
 - Amenorrhea + symptoms of early pregnancy
 - Bleeding → moderate or severe
 - Pain → marked lower abdominal colicky pain with backache followed by expulsion of the conceptus ⇒ 5 bleeding & pain
- > Signs General → according to amount of bleeding my be slocked.
 - Uterus (bimanual) → smaller than period of amenorrhea
 - Cervix $(P/V) \rightarrow closed$
- > Investigation U/S → empty uterus
- > Treatment 1. Echolics ± Antibiotics Resuscitation.

 2. However, some do → DaC (so ensure complete evacuation & line 3-Anti-DIF Rh-ve.

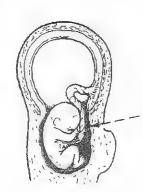
4) Incomplete abortion

Symptoms, Signs

As inevitable abortion but part of the products of conception have been expelled \rightarrow therefore the uterus is < period of amenorrhea (confirm by US)

> Treatment → as for inevitable abortion

Cervical abortions: Contraction sisable to open The internal of but not external os.



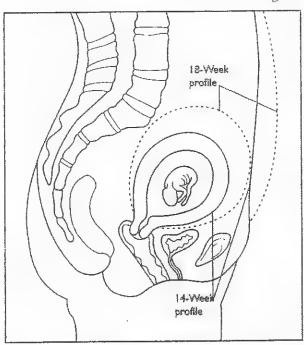
-Normally high E - setimulate Athitary -> 1 Proloction
- Fibringen (end -> Normally -> 200-7.00 mg/dl.
- CX. preg. Sung Preg. -> 400-600 mg/dl.

- DIC, has very strange chich!

1-Take very long time (4-6 w), only exception

2-The only Case of DIC that can be TIT by

Leparin.



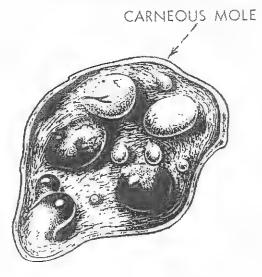
Missed abortion. The duration of the pregnancy is 18 weeks but the uterus has failed to enlarge beyond the size of a 14-week gestation. Note that the abdomen is flat.

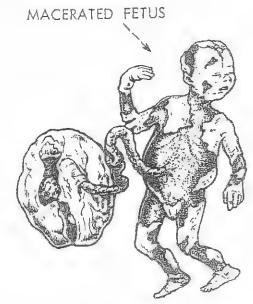
A Fresh blood: < 8 hours.
-EUSA for HCV, HIV take 20 hours.
- so Fresh blood show nove liability to
transmitt infection.
-Inspite of that fresh blood is much
-Inspite of that fresh blood is much
used Scorrects hypovolenia.

Atterain Con berned in Case of single Introvite ine fetal death in atwin-s Prophylaxis against DIC to stop The Wiscons Circle.

Then to use preparane 2-No ble eding. 3-No of exactions.

If retained for long enough, the gestation may end up as a





Cervical abortion

	Cervical abortion	Cervical preg. (v.rare)
Def	type of inevitable abortion	type of ectopic pregnancy
	→ arrest of gestational sac	→ implantation in the
	in the cervical canal	endocervical canal
Sympt.	pain (severe) > bleeding	bleeding > pain = nofain dutor a d. al dit External os opened
Signs	Internal os opened	External os opened
iii	Dilatation & curettage	Hysterectomy OR conservative
when the devel of the same throughout the same property	and Appent of the property of the Control of the Co	NOC 10 FLOO

(Carneous mole...fleshy mole...bloody mole)

Definition retention of dead / non-viable products of conception within uterus

Paragene us: Spreadof Wood - Noseritation of the where so Nogain

Sumptoms Use of Projectione - masking of symptoms

1- Amenorrhea = symptoms of pregnancy disappear - N. and

2- Bleeding = rarely mild dark brown (prune juice) Milk secretion * (d.t. ♥ E)...it may occur normally in preg.

. Normally E2 blocks action of prolactin on breasts in preg. (Receptor be

3- Pain susually absent + absent fetal movements

> Jigns

- * No general signs of pregnancy
- * Uterus → less than period of amenorrhea
- * Cervix -> closed firm ± dark brown discharge *

Investigations

- 1- *Ultrasound* → collapsed pregnancy sac + no fetal pulsations
- 2- B-HCG & repeat in two days for doubling /-ve or no doubling).
- 3- Fibrinogen level (very important) as there may be liberation of thromboplastin substances from the retained dead tissue which may lead to DIC SLOWLY. In these cases fibringen level usually decreases by 50 mg/ week. : it is done weekly to avoid reaching the dangerous level (100 mg/dl)

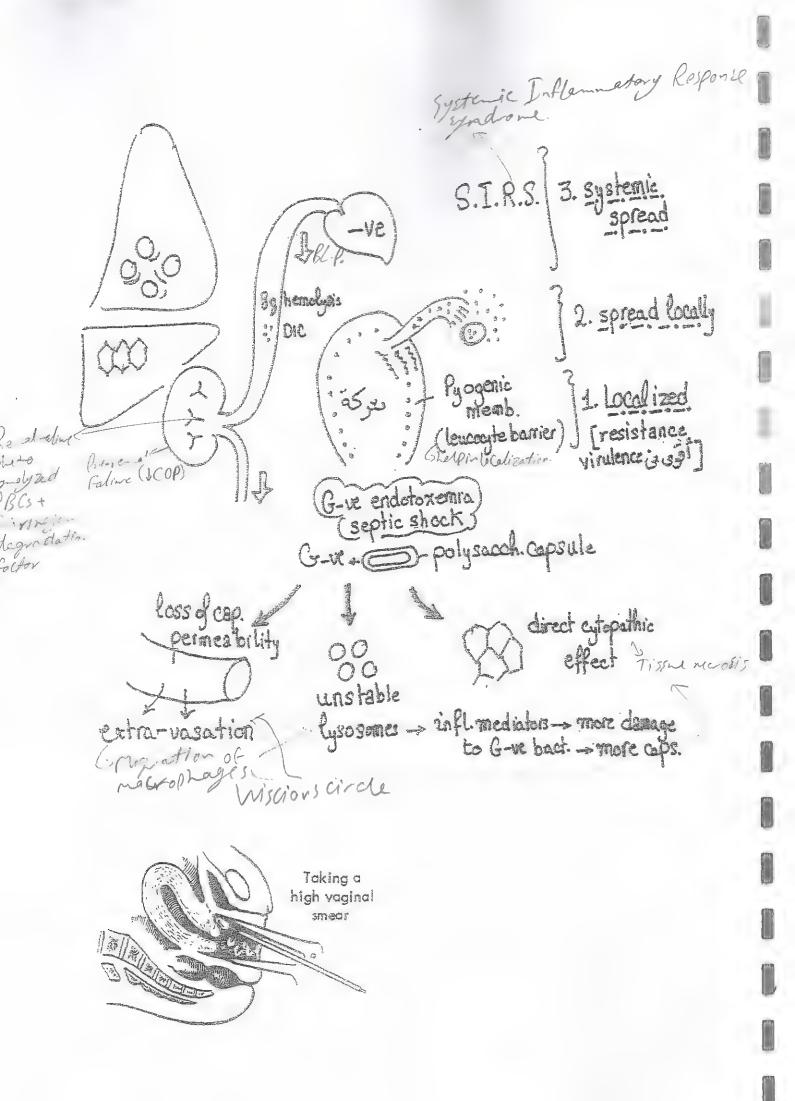
Complications

Infection -> septic abortion

DIC (hypofibrinogenemia) → after 4–6 weeks

less 14 w. -> Dand C or waston Treatment Teminetion of Damere Inw. secbolics If fibringen is normal = TOP (acc. to gestational age) + antibiotics

If fibrirogen is $\Phi_{\rm cd} =$ elevate 1st (fibringen, FFP, fresh blood) then TOP CYTOPPT



7A Saelitanierition

commendation is Considered septic

Definition superimposed infection on any type of abertion (esp. crimmal)

Organisms

- Gram +ve → Staph, Strept. esp Group B (GBS)
- Gram -ve → E.coli, Pseudomonas
- Anaerobic → anaerobic Strept, Bacteroides, clostridium epreviously

> Source:

- Exogenous.....instruments, sanitary oads
- Endogenous......organisms present in temale genital tract
- Hematogenous (rare)..... from a septic focus e.g. appendicitis

Clinical Picture

Sumptoms

- * Symptoms of abortion (amenorthea...bleeding ...puin)
- Followed by symptoms of infection
 - Fever, headache, anorexia, malaise, rigors
 - Continuous lower abdominal pain
 - Offensive discharge
- * There may be history of a tird to induce abornously. By the income has

Signs

- 1- General → Toxio, pale, tachycardia, tachypnea, high foliair
- 2- Abdominal
 - Decreased abdominal provement with respirance spilos
 - Lower abdominal tendemess & rigidity
 - Tender utorus may be felt

3- Vaginal

- Bleeding & offensive discharge
- Uterus is tender rurely crepitations it interest with gas

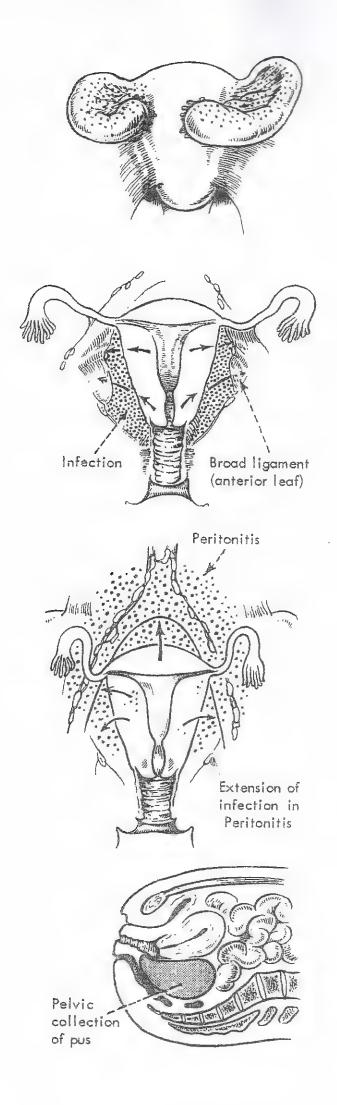
- Swelling in Douglas pouch - peis to absect

Investigations usually not needed, clinically diagnosed.

- I FOR PIAGNOSIS: U/S → dead fetus or incomi iere nbc."
- 2. FOR FROLOGY
 - Blood ELLTURE (TLC, ESR. CR3) Aarte Place reactents.
 - Endocer real or high vaginal swaps
 - X-ray physometra gas in uterus + gasunder diaptragm.
 - FOR COMPLICATIONS > rena. include test & coagulation profile 6601 en 100

RF

DIC



	STRS)L
Complications Φ	Jo Hf due to Cardiotox	icity
Local General	Organ affection	6
1- Endometritis 1- Septic	1- Septic shock, ARDS → R-&P. f.	
2- Myometritis thrombophlebitis	2- Acute haemolysis	
3- Salpingitis 2- Systemic	(esp strept & clostr) + liver	
4- Salpingoophritis pyaemia	affection → jaundice 3-DIC 4-Renal failure due to : kingendage the above factors.	: Pre s
5- Parametritis 3- Generalized	3-DIC adme to 5- himselv 200	IFBG.
6- Pelvic peritonitis peritonitis	4- Renal failure due to stringe dags	-adatio
7- Pelvic abscess	the above factors. "chict -> Re	model a
A Third and the same of the sa		100
Treatment		Bleeding
1) Elevation of the general condition		<u>a</u> .
Antibiotics (in combination in high doses)		g
- $Gram + ve \rightarrow penicillin G or cephal$	losporins	-
- Gram-ve → aminoglycoside as gen	tamycin or tobramycin uss replication	G O
		2
- In gas gangrene → specific antiseru		Y
Close observation in the ICU (in complicated))re
- Vital data → blood pressure, pulse	· ±	gn
- CVP esp. in renal affection → . Un		Q
	epeated renal function tests	early Pregnancy
- Blood transfusion (better fresh) an		
- Hydrocortisone or dexamethasone		
. † tissue perfusion, stabilize	: rysosomes & endomenum	1
	e sensitivity to catecholamines	TON.
2) Evacuation of contents when ofter		
1 st trimester suction evacuation (bette		
T.	of infection by opening sinuses	
_	tion of the soft uterus	
2 nd trimester * induction of abortion	n by oxytocin or PG	/1
weak wtern " if failed → hysterote	21,000	1
	systerectomy -in toto- (esp. if old,	
	er family, gas forming organisms)	
3) Treat complications		
1- Pelvic abscess → posterior co	olpotomy	
2- Septic thrombophlebitis → heparin		
3- Generalized peritonitis → drainage		

→ fibrinogen, FFP, fresh blood

5- Circulatory collapse → vaso-pressors & sympathomimetic drugs

→ assisted ventilation

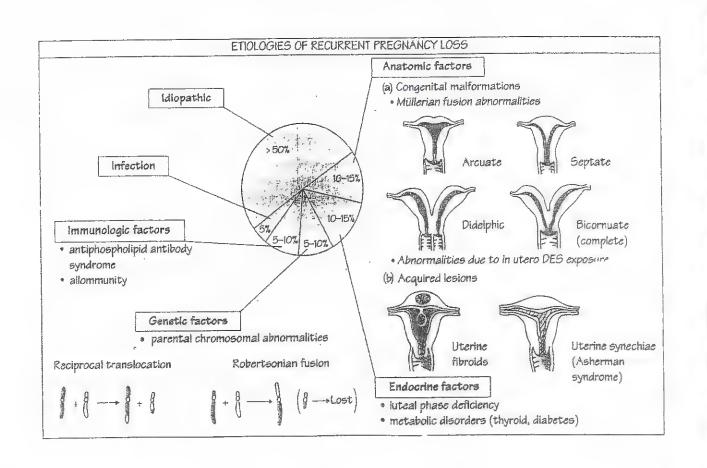
→ dialysis

4-DIC

7-RDS

6- Renal failure

Atterine hypoplasia. a bortion in Ascendir in it is entering it is



8) Habitual / recurrent abortion

Definition

Three or more <u>successive</u> spontaneous abortions (some say *two*) (If not successive it is called <u>repeated</u> abortions)

Incidence

- \Box **PG** \rightarrow 10%, then percentage depends upon previous abortions:
- \Box *Once* \rightarrow 20%
- \Box *Twice* \rightarrow 26% (2–3 % of community)
- □ *Thrice* \rightarrow 32% (< 1% of community)^{π}

Of Progration of Aspirin

Of Folic A.

1 Local causes

- Account to 30% of 2nd trimesteric abortions
- Most of them is not diagnosed before multiple pregnancy losses have occurred (because they are .asymptomatic)
 - 1] Patulous internal os
 - 2] CMF of uterus → septate (25%) or bicornuate (30%)
 - 3] Uterine hypoplasia -> abortion in ascending manner
 - 4] Submucous fibroid
 - 5| Fixed RVF → abortion usually at 14-16 weeks when
 - 6] Congenital Asherman syndrome \rightarrow intrauterine synechiae

General causes

- 1- Endocrinal DPD....PCO.....DM......Thyroid
- 2- Immunological -->
 - * Autoimmune \rightarrow APS $\checkmark\checkmark\checkmark$, SLE
 - * Alloimmune -> . RH incompatibility
 - . Excessive HLA sharing
- 3- Thrombophilia → hypercoaguable state: ↓ protein C_&S or AT₃ factor V Leiden ↓, hyper-homocysteinemia, Ptn Z ↓
- 4- Infections e.g. toxoplasma (recently not believed to be a cause)

Fefal (genefic): 4–10% " (structural anomalies)

- * Translocation, inversion
- * Mosaicism, deletion

1 Hanna

> Personal

- û Age → chromosomal anomalies, DM, hypertension
- Residence → rural areas (Bilharziasis), slum areas (toxoplasmosis)
- Occupation → workers in heavy metal or radiation factories
- ~ Complaint abortion > 3 (2) times

> HPI

- Symptoms of abortion → amenorrhea, bleeding, pain
- Symptoms of complication → fever, DIC

Past

- Medical → hypertension, DM, thyroid, heart disease, ...
- Surgery → on cervix: trauma

-Family

- Diseases → hypertension, DM,

>Mensirual

- Premenstrual spotting \rightarrow LPD
- Menorrhagia → fibroid
- *Hypomenorrhea* → hypoplastic uterus, Asherman S

> Obstatric

- $Timing \rightarrow 1^{st}$ trimester is usually due to 2^{nd} trimester is usually due to
- Order → Ascending.....

Descending.....

- Character of abortus → Fresh.....
 - Macerated.....
 - Malformed.....
- Special $C/P \rightarrow \text{in PIO}$ (painless, smooth, rapid, easy)

2 Examination

- > General medical disease
- > Abdominal swelling: fibroid uterus, ovarian swelling
- > <u>Local</u> -
 - Uterus. Small (hypoplastic)
 - RVF
 - Bicornuate (2 bodies)
 - Carvix: short, tear (PIO)

AHSG is more accuracy in a agnoss of where amendies of U/S.
A Toxplosmosisgines solid immunity -> cause abortion for one time only.

Management

Cause	Investigation	Treatment
Patulous os 🗸		and a supplied to the supplied of the supplied
Septate uterus	 If pregnant Ultrasound better transvaginal 	Metroplasty (only if there are repeated failures of vaginal cerclage) as it leads to extensive adhesions •
Uterine hypoplasia Submucous fibroid Fixed RVF	 If not pregnant ➤ - HSG - Hysteroscope 	Cyclic estrogen & progest.
* LPD * PCO * PCO * DM * Thyroid 2- Immunological * APS * * SLE * RH incompat. * HLA sharing 3- Thrombophilia 15 % 4- Infections	 progesterone ↑ LH & androgens GTT T₃, 4 TSH PTT, anti-CL, LA C_{3 & 4}, ANA Rh titre HLA typing Screen for protein C_{&}S or AT₃ 	Progesterone (100 mg 1x2)Induction of ovulationInsulinL- thyroxinelow dose aspirin + heparinsteroidsaccording to titreimmunotherapy: blocking abdsLow molecular weight heparin → anticoagulationSpecific ttt acc to C&S
Genetic factors	 Family history Karyotyping of both parents (or abortus) 	CounselingDonor gametes (unethical)

**** If no cause is found (very common >50%)

- Reassurance, more periods of rest, avoid exhausting trips
- Good diet, vitamins & iron, stop smoking & alcohol
- Empirical drugs 👄
 - Folic acid (3 months < & > preg. → \ neural tube defects) \ CNS anamalies.
 - . Progesterone, low dose aspirin, heparin

Notes

CX dilatation: 1 inwidth. CX effacement: I in long the

> Etiology Ф

- Congenital
 - Increased muscle tissue in cervix > 10%
 - Associated with other uterine malformations as septate, bicornuate uterus, hypoplastic uterus
 - DES (diethyl-stilbesterol) exposure in utero
- Acquired
 - * due to obstetric trauma
 - Forceps or ventouse before full cervical dilatation
 - Breech extraction before full cervical dilatation
 - Manual dilatation of the cervix
 - * due to gynecologic trauma
 - Dilatation of the cervix excessively or too rapidly/ \(\mathcal{L} \)
 - High amputation of the cervix
 - Cone biopsy of the cervix

> Clinical picture (---, Hispy) (ci-

- This condition usually leads to classic picture of (6-----)
 - Painless effacement & dilatation of the cervix
 - Uterine contractions are late & not very painful
 - PROM followed by rapid delivery of a fresh abortus with minimal discomfort
- The abortion or premature labor usually occurs in <u>descending</u> lover limit is fashion i.e. at 7 months \rightarrow 6 months \rightarrow 4 months at a fashion i.e. at 7 months \rightarrow 6 months \rightarrow 4 months, etc.

> Investigations No med of inv.

1] If pregnant

- Serial U/S examination (better done transvaginally) - to determine length (2.5-3 cm) & width (1 cm) of internal os

2] If not pregnant

- HSG → funneling (loss of uterine waist)
- Ability to pass Hegar dilator No 8 or hysteroscope No 8

 with no resistance & little pain X

 Pediatric Foley catheter with 1 ml inflated balloon can be
- Pediatric Foley catheter with 1 ml inflated balloon can be pulled through the os without resistance x

> Treatment

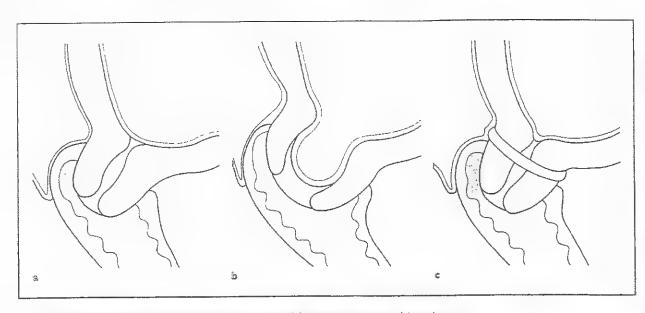
1] If pregnant \Rightarrow cerclage

Til. To, stable

Bleeding in early Pregnancy

3 40 11 M 3 2 - 3 16 X Sporo.

CS afte Co clage: 1- Abdominal corclage.



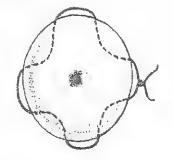
Cervical incompetence. (a) Normal cervix at 16 weeks; (b) Incompetent cervix at 16 weeks; € 5. 6 em 1 15 10 00 W

(c) Cerclage with an unabsorbable suture. already started c ft i eb. - PROM 1-Patrolysiology has

9 eVithal Shirodkar (1899-1971) was an obstetrician and gynaecologist from Goa who proposed a pursestring suture of fascia lata around an incompetent cervical os to prevent second-trimester loss.

Ian McDonald (1922-1990), from Australia, simplified the Shirodkar operation with the use of a silk purse-string suture around the cervix.

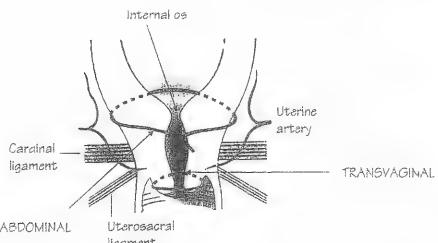
1-Patholhysicion has not toan ted yet 2-Per Certage of spontanous abor Is high in let trimecter.



. Avoid 3 & 9 o'clock > to ovoid injunt of descending can covaginata.

Ends of tape are left

for easy removations, hanging from post. lip of cx



TRANSABDOMINAL

ligament

Bleeding in early Pregnanc's

> Tupes

1. Meteraids → 70% success

. 4 bites with purse string suture (Nylon or Mersilene)

. Taken around the highest portion of the portio-vaginalis...why? as wear as fossible

2. Modified Shirodkar's → 70% success

. The bladder is dissected upwards

. Thus sutures could be taken at level of internal os

3. Abdominal -> at level of internal of -more strong.

. Indications → repeated failed vaginal cerclage or short / absent cervix

. Delivered by \rightarrow CS (permanent cerclage)

. If failed ≤ 28 wks \rightarrow hysterotomy must be done (a great disadvantage) Gthe weakest scar in the uterus.

Indication \Rightarrow PIO, uterine malformation (septate), triplets

- 78 Coly + C- to preve + weile C.d. Postoperative = anti-PG, progesterone, antibiotics, Beginnist-

Removal \$\Rightarrow\$ 2 weeks < EDD (\$\approx 37 wks) >> before start of utasi.

Complications

- Injury to bladder

- Injury to membrane → ROM → tape must be removed funder any Condi

- Infection → tape must be removed & terminate

- Abortion or PTL -> dul to irritation.

Antiphospholipid syndrome

▶ Definition . autoimmune dis. forming antibodies against phospholipid proteins . it may be 1^{ry} (alone) or 2^{ry} (associated with CT disorders: SLE)

➤ Diagnosed by ΦΦ

O Recurrent ""

and to formation of Abs against intermed of BVs.

- Thrombosis → arterial & venous

- Fetal loss $\rightarrow . \ge 3$ consecutive miscarriages (< 10 wks)

 $. \ge 1$ fetal death (> 10 wks)

. ≥ PTL (< 34 wks) due to severe PET

- PIH → usually severe ± pl. insufficiency ± IUGR ± abruptio pl.

Positive antibodies

- Anticardiolipin antibodies (ACA) } high

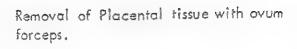
- Lupus anticoagulant (LAC) } false +ve

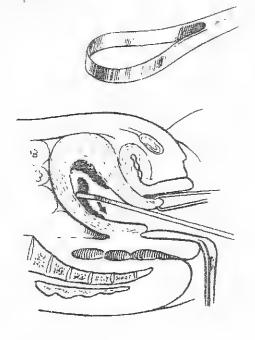
> Treatment

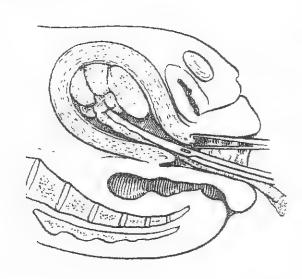
* Baby aspirin (75 mg/dav) Heparin 5.000 units SC /12 hrs or LMW heparin / 30-40 mg /day >

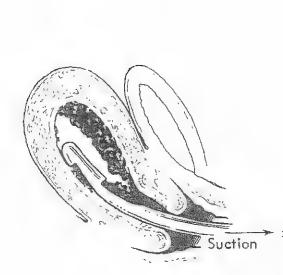
Corticosteroids → not more used

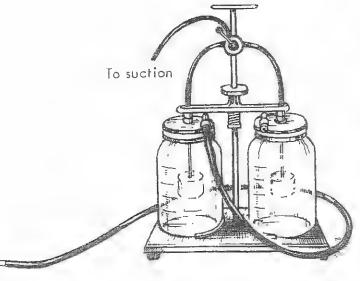
CURETTAGE. A blunt curette may be tried first but usually a sharp curette is required.

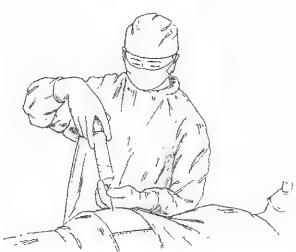


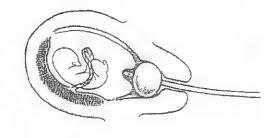












Ingection of hypprtonic sonution

Local PG (Intrauterine-Extraamniotic)

@----- Induced abortion-----

Therapeutic abortion

> Indications

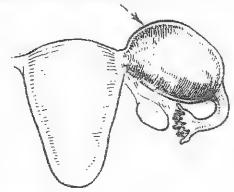
- 1 Maternal
 - Medical disorders e.g.
 - Advanced: Heart disease / chronic HTN / renal disease
 - Active pulmonary T.B./ severe hyperemesis
 - o Malignancy
 - Genital tract / breast malignancy
 - Chemotherapy or radiotherapy
 - Mental psychological illness
- 2 Fetal
 - o Missed abortion / blighted ovum / vesicular mole
 - Exposure to teratogenic agents → rubella radiation
- ▶ Methods
 - A- Before 14 weeks → suction evacuation Or dilatation & curettage
 - B- After 14 weeks
 - Prostaglandins
 - Local (intra-amniotic or extra-amniotic)
 - Vaginal or intracervical tablets
 - o Oxytocin
 - Intra-amniotic injection of hypertonic solutions 🗶
 - Saline 20%.....Urea 30-40%......Glucose 50%
 - Complications → danger of infection & DIC
 - o Hysterotomy if all fail or there is severe bleeding

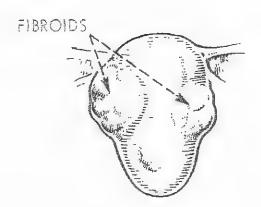
2) Criminal abortion

- ▶ **Definition**: TOP for non-medical reasons (in countries where abortion is illegal)
 It is called elective (voluntary) abortion (in countries where abortion is legalized)
- ▶ Methods used
 - 1- uterine stimulation → methergine, purgatives
 - 2- Intra-uterine manipulation to induce cervical dilatation or ROM
 - 3- Evacuation by untrained Doctor under Septic conditions
- Common complications
 - Genital tract trauma e.g. uterine perforation
 - Infection → *sepsis*
- ➤ C/P & treatment as SEPTIC abortion

ABORTION-DIFFERENTIAL DIAGNOSIS

TUBAL PREGNANCY





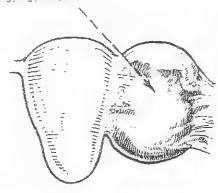
METROPATHIA HAEMORPHAGICA may simulate abortion so crossly that the distinction can be made only on the histological appearances.

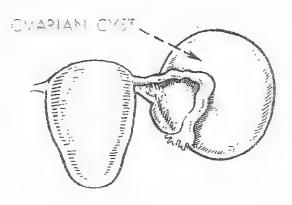
- scientefic tem.

- induced - still 2: Loss.

- 1st trimertest - sego jes relo

9703AE9117





Much prefine bleeding has no pregants explanation and the patient accepts or supplies a diagnosis of abortion for want of anything better.

- lay people term.
- spontaners e skil l'I, of ex
- 2nd trime et er of l'ele

KEYPOINTS

- 1. The most common cause of first trimester abortions is fetal chromosomal abnormalities.
- 2. It is important to rule out ectopic pregnancy with history, physical examination, laboratory studies, and ultrasound.
- First trimester incomplete, inevitable, and missed abortions are usually completed with a D&C or medical management with prostaglandins, although expectant management is also used.
- 4. RhoGAM should be given to all Rh-negative patients with bleeding.

KEYPOINTS

- Most second-trimester abortions are secondary to uterine or cervical abnormalities, trauma, systemic disease, or infection.
- D&E, prostaglandins, or oxytocic agents can be used for the management of spontaneous abortions in the second trimester that need assistance to completion.
- 3. The risk of uterine perforation from D&E is greater in the second trimester than in the first.

	The state of the s
>	What is medical abortion?
>	What is the DD of abortion. ? Look the figure ectair, voicular selection of the blooding & Blooding
	What are the complications of abortions. The Long sequelae
, Cer	tion brock blertoration bashernans.
3	What are the causes of postabortive bleeding? THE COMMONESTTHE MOST SERIOUS PERFORATIONASSOCIATION THE PROPERTY HAVE
21	THE COMMONESTTHE MOST SERIOUSPERFORATIONASSOCIATION
· fa	torn Hge
A	What is the management of postabortive bleeding?
>	What is the weight of the smallest fetus ever survived? 375 gm· What is the difference between term abortion / miscarriage? What is the management of a case of idiopathic habitual abortion?
*	What is the difference between term abortion / miscarriage?
*	What is the management of a case of idiopathic habitual abortion?
· San	How to prevent spontaneous abortions?
	Most abortions could not be prevented (except if there is obvious cause e.g. DM, PIO). This seems reasonable as most spont abortions are due to C.F M.F.
	What are the other rure causes of bleeding in early pregnancy?
	LOCAL GYNECOLOGICAL CAUSES Known by speculum examination e.g. ulcer, polyp, HPV, tumor
	THARTMAN'S SIGN (scanty spotting at time of implantation)
	- Due to erosions of some vessels 1 week after fertilization (during infebrutary
1	- Importance → wrong calculations of EDD DECIDUAL haemorrhage (monthly scanty <u>bleeding</u> at time of menstruation)
Ò.	- Due to separation between decidua capsularis & decidua parietalis
Ú	- bleeding occurs till 12 weeks (until the 2 layers fuse together)
>	What are the main etiological causes of the 2 nd trimesteric abortion?
	* Cervical incompetence & other uterine malformations

* Early fetal demise (early IUFD) e.g. syphilis, Rh (both rare now)

* <u>Placental</u> causes e.g. APS, circumvallate placentation * <u>Uterine</u> overdistension e.g. twins, acute polyhydramnios

* Trauma & infection

Effect in DES on & genital system:

1-Vaired admosis -> adeno Concinoma.

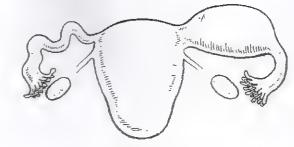
2-fatulous int. OS. -> hollowed abortion.

3. T-shaped wtens

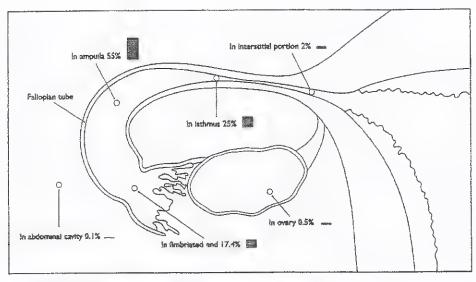
U. G. getial tubal anomalies.

U. G. getial tubal anomalies.

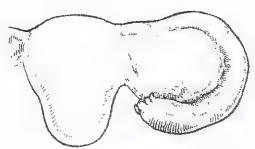
Chypoflosia, accessory oction, diverticula.



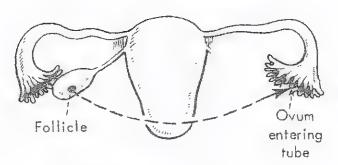
Tubal pregnancy

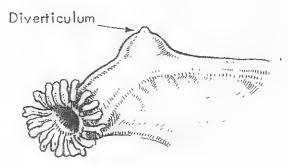


Sates of eccepic gestation implantation, with the relative frequency of occurrence.



Chronic salpingitis





Protectine (and vulati

Ectopic Pregnancy

Definition

- Implantation anywhere outside the <u>Endometrial Cavity</u>
- It is responsible for 10% of MMR
- INCIDENCE is ↑^{ed} 4 folds in the last 20 yrs from 1→3 % d.t. ↑ of: *
 STD's ② contraception (IUCD) ③ ART (IVF)

Sites

Uterine (v.rare)	*** Charles Charles (Security Security Security Security Charles and Confinence Charles Charle	<i>xtra-uteri</i>	ne	Described framen, we (CEE PERSONAL CENTRAL OF SPEEDERS - FASTAL OF
1- Cervical	1- Tubal 99%	Name of the second of the seco	The-	nost vide
2- Rudimentary horn	Interstitial,	isthmus,	ampulla,	fimbria
3- Angular = Grandar 4- Intraligamentary > Broad by.	2	15	√ 80 ¤	5
4-Intraligamentary	2- Ovarian 0.5	%		
Sproadly.	3- Peritoneal (a	bdominal	1^{ry} or 2^{ry})	(4). Insumations/orders_adops_proximals 18 albei 1904 (1914)

Etiology $\Phi\Phi$

1 Causes in the tube preventing normal transport (mechanical factors) -> delayed in plantation

- Congenital > hypoplasia, accessory ostia, diverticula
- Traumatic surgery on tube.....tuboplasty, tubal ligation or surgery near tube.....ovary, uterus, appendix
- Inflammatory 1 50% = PID (chlamydia > gonorrhea), appendicitis
 PID=SID=schlamydia. \(\) peritubal adhesions (esp on the right side)
- Neoplastic = tumors in broad ligament, ovary, uterus

 stretch of the tube & obstruction of ostia

Miscellaneous

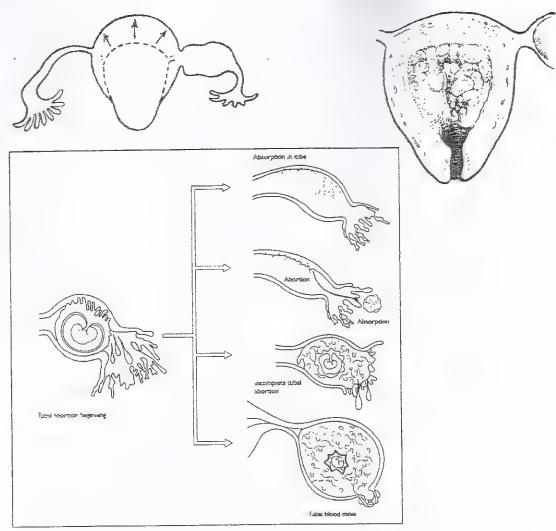
- Endometriosis adhesions deto bleding.
- ART → ↑ ectopic by 5% (due to

- Contraception:

- . POP or Implants $\rightarrow \mathbb{Q}$ tubal motility (what about COC^{π} ?)
- . IUCD → salpingitis, ↓ tubal motility (esp if + P), also it can prevent intra- but not extra-uterine pregnancy for by IUCD → sectoric be Court + boverall freg. rate.

② Causes in the fertilized ovum

- Early disappearance of zona pellucida
- Early development of trophoblast
- External or internal migration (time consumption) X



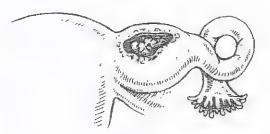
Sequence of tubal abartion.

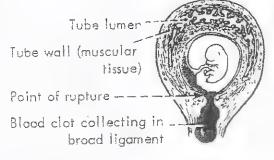
RUPTURE INTO LUMEN OF TUBE (TUBAL ABORTION)





RUPTURE INTO THE PERITONEAL CAVITY





Sometimes rupture is retroperitoneal between the leaves of the broad ligament - broad ligament haematoma.

Broad ligamentary hematoma

Boby rorely = 2" intra-ligamentary pregnancy

Gyming 11. 101 sterrician must be expected winder.

Any lieg. shouldbe Considered exteric + U proved otherwise.

Twins

Symptomisign	Ectopic gestation (%)
Abdominal pain	90
Amenorrhoea	80
Adnexal tenderness	80
Abdominal tenderness	80
Vaginal bleeding	70
Adnexal mass	50

Asymptomatre->ifdiagnosed > best chance in TII الرين المري (2%) 1. Undisturbed ectopic (2%) SymptomsTRIAD the most imp. is pain - • Amenorrhea (short period) + symptoms of early pregnancy Pain → slight dull aching in one iliac fossa (tubal stretch) واحت كالرم Bleeding → usually absent or slight spotting Signs - General → signs of pregnancy - Uterus → soft, slightly enlarged, y wetn'ce! - Adenexae → . slight tenderness in one fornix and for Clof Preg. Bleeding in early Pregnancy . sometimes a swelling may be palpable (< 3cm) Early diagnosis needs HIGH LEVEL OF SUSPICION - There is much need for - History of pdf (e.g. PID, IUCD) + you must be ectopically minded - May be discovered accidentally during routine U/S of pregnancy مريث من يه 2. Subacutely disturbed ectopic (60%//) acrute abolomen + Int. Hye. Symptoms AMENORRHEA: (may be mis diagnosed e' men et mad ir regularitées) - Short period 6 – 8 wks - Mostly there is one missed period Sudden severe PAIN: Dull aching → tubal distension - Sharp stabbing → erosion through the wall+ Peritorean - Colicky → tubal contractions (tubal abortion) Si Vaginal BLEEDING: Few days later, , very wild. - Drop of β-HCG → I E & P → separation of decidua → 1th drawed bleeding. - Slight dark brown (or rarely as a decidual cast) Signs > General - Various degrees of shock → coma in severe cases) - û pulse, ♥ BP, ♥ temp., cold clammy skin, oliguria Abdominal

Tenderuss - Jed movement of lower abdomen with respiration Rigidity - T, R, RT over lower abdomen

- □ Cervix = extreme tenderness on movement JUMPING sign
 = cervical motion tenderness // Texterness of two.
- Uterus difficult to palpate (tenderness) but is slightly enlarged

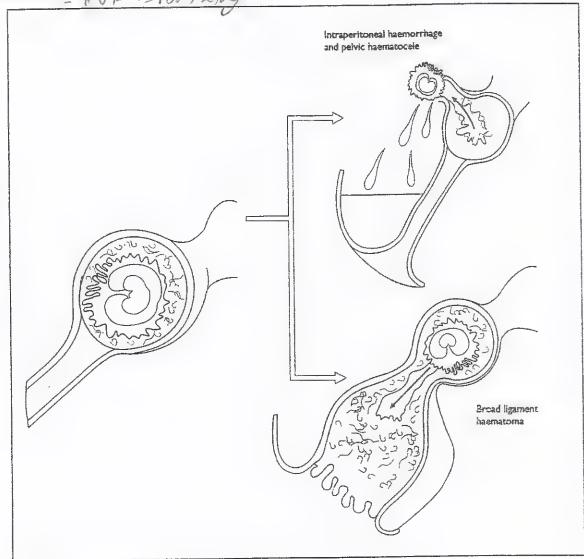
□ Adenexae = tender enlargement of the affected adir cum +5 celling.

hematoma. Welseli's on 30 dr

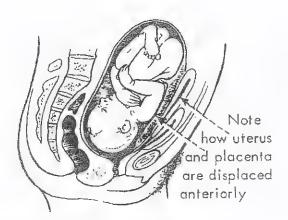
The Commonest suching in D. Pouch is RVF uterus 20%.

1-Biraval ex: RVF-suterus is not felt i herato Cole - Cx Pushed A. I, directed down 2-PV: RVF-> Cx directed forward i herato Cole -> Cx Pushed A. I, directed down 3-Culdo Centeris: - herato Cele -> Blood.

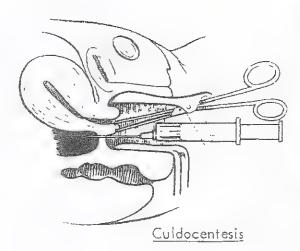
3-Culdo Centeris: - herato Cele -> No thing.



Sequelae of tubal rupture.



ABDOMINAL PREGNANCY



DD: 1-RVF weres. 2- Postwall fibroid.

3. Acute (fulminating) type (id) worked for

Symptoms

..... Short period of amenorrhea -> sudden severe abdominal pain

...Followed by: massive intraperitoneal hge with shock & collapse ± shoulder pain: diaphragmatic irritation by blood

Signs

➤ General → shock (not proportional to external hge)

Abdominal

> Vaginal → difficult (marked tendernoss)

4. Chronic pelvic haematocoele

Symptoms

.....There is history suggestive of disturbed ectopic preg (the triad)

.....Then blood collects gradually in the D.pouch (most dependent)

....Leading to pressure symptoms (backache, dysuria, dyschazia, dyspareunia)

Signs

➤ Vaginal ⇒ tender ill-defined boggy mass in D. pouch pushing cx anteriorly

Que , lelo de so o

Treatment..... evacuation by

1- Laparotomy + strong antibiotics

2- Posterior colpotomy (or aspiration guided by TVUS)

1. V V.V. YEVE Advanced abdominal pregnancy

Signs

 \rightarrow Abdominal \rightarrow . Abnormal lie (e.g. high transverse lie)

. Easy palpation of fetal parts

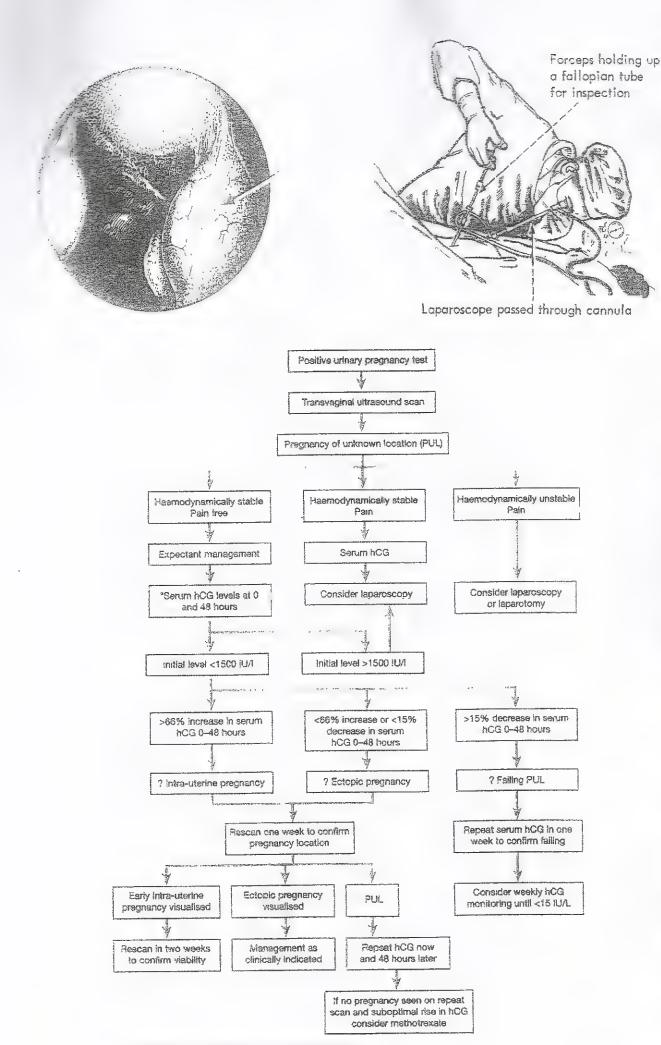
> Vaginal → uterus small & separate from fetus

Investig. = U/S (CT, MRI):- fetus & placenta are outside uterine cavity fray (at: Vew ove laborgof fetaland natural skeleton. Greatment...... Laparotomy

- 1- Laparotomy \rightarrow remove fetus & sac (fetus is malformed in >50%)
- 2- Regarding placenta:
 - If attached to unimportant structure as omentum → remove it
 - If attached to important structure or great vessels →

.Cut the cord short & leave placenta for absorption

This takes 1-2 years (methotrexate may help absorption)



Algorithm for managing suspected actopic prognancy.

after 66 eding, -> 6600d

hashot yet clotted.

(Investigations)

l Pregnancy test

- Serum β-HCG ✓ (detects 5 mIU/ml) is more sensitive than urine
- intrauterine preg. = normally doubles 12-3 days
- Estopic preg. subnormal rise: less than 66% within 2 days

(But it may be non-viable intrauterine pregnancy)

GThreatened or rissed abortim. 2] Ultrasound - diagnosed on freence of enft whens lexchasion of where Preg

- Vaginal U/S is more sensitive than abdominal U/S
- Intrauterine preg. ⇒ gestational sac in-utero (5 wks TV..... 7 wks TA) (But it may be the decidual reaction of ectopic preg)
- Ectopic preg ⇒ a small sac ± fetal echoes outside the uterus (But it may be CL cyst of normal preg)

If diagnosis of intraperitoneal hge is evident -> proceed directly to laparotomy. Otherwise: in early undisturbed cases

ightarrow do the following (as diagnosis is difficult/ query)

Hospitalization & follow up of

- * <u>Symptoms</u> → pain
- * Signs → detectable adnexal swelling
- * Investigations → /2 days

discrimination Combined U/S + B-HCG) powiff The discrimination value at which U/S can detect an intrauterine pregnancy is:-

- 6.000 mIU/ml (by abdominal U/S), or

- 2.000 mIU/ml (by vaginal probe)

Any level above this + no intrauterine preg. detected by $U/S \rightarrow will$ be most probably ectopic fresh blood - Just

Serial Hb & Hct)

Internal hge is suggested by the progressive

drop in Hct in absence of external bleeding If diagnosis is still query of the misdiagnosed (4-8 %) -> small fet w. If blood is Clott ed -

* Laparoscopy → both diagnostic & therapeutic 🗸 🗸

Culdocentesis -> tapping of blood from D. pouch x after bleeding -> cletting in D. Pouch -> liquified by fibrinsbytic system = No mare clotti.

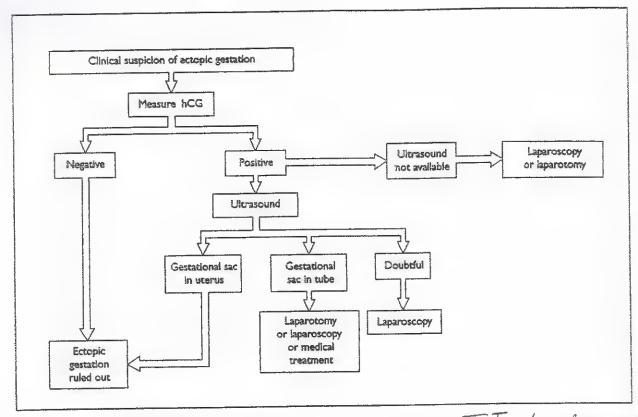
» Progesterone level

 $a \ge 25 \text{ ng/ml} \rightarrow \text{normal intrauterine pregnancy}$

5 ≤ 5 ng /ml -> abnormai (ecropic or non-visible intrauterine preg.

DaC XX => decidua but no villi it may disturb an early healthy pregue yEUA tu => it may increase ti nubance

EX. under Ansesthesh.



Algorithm for diagnosis of suspected ectopic gestation.

① Ecfolic.

DD'② PTD: Usually bilat. + feve + No an ever hear

③ Per sistent CL!

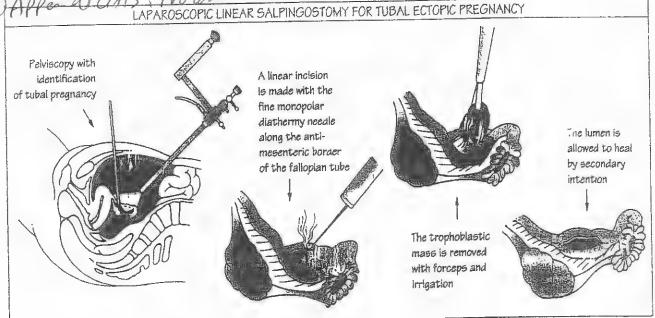
① Ruthered over lan cyst! No an ever hear.

⑤ Fibroid. (Rat degeneration).

⑥ After di Citis! No an ever hea.

⑥ After di Citis! No an ever hear.

Chaparoscopic Linear Salpingostomy for Tubal Ectopic Pregnancy



(Treatment)

□ Resuscitation \/ \/ \/ → anti-shock measures

الا wide bore cannula + call 4 help

- Labarotomy (or laparoscopy)
 - Peritoneal toilet → to remove blood
 - 1st inspect the other tube (may be diseased, absent, malformed)

• Salpingectomy → affected tube is removed / (the best; to avoid ____) Reccurrence. Oble eding dect + . Oophrectomy *x = to force the other ovary to ovulate monthly 2. No oophrectomy hormone production only theoriti adnot 10 occurry Practically 0 Conservative surgery if one tube is present or } every attempt mild cases or Just a mail should be done to conserve the tube in early Pregnancy as it may Course recurrent ectors low parity

(6), some fefere to do
This is in the form of linear incision (at the anti-mesenteric border)

Salpigotomy: tube is closed by → sutures

. <u>Salpingostomy</u>: tube is left open → heal by 2^{ry} intention as intures

. Partial salpingectomy (never to be done).....high recurrence x Milking the tubal contents (esp if near fimbria)..... the worst x

ny cousa-severe adresion.

- Laparoscopy (diagnostic & therapeutic)
 - Same procedures as in laparotomy may be done, but needs:
 - Expert team + special equipment + haemodynamic stability (not shocked ") is in disturped actoric.

• Adv. \rightarrow done as a day case

distiked by many leaple. Medical fit (conservative to fertility)

• Methods . Methotrexate folic antagonist (IM 50 mg/m²)....once Misepristone (RU-486) $\mathcal{P}G$ - $\mathcal{F}_{2\alpha}$ (locally in the sac).....laparoscopic or U/S guided

• Criteria = . Sac size < 3 cm, -ve cardiac activity (non-viable) 5 Sweeks. . β-HCG < 3000 mIU/ml

disturbed) . Patient haemodynamically stable

Follow up
 . serial Hb & Hct levels, TVUS, β-HCG

sually I do ses are needed door is reported in the starts (day 4)

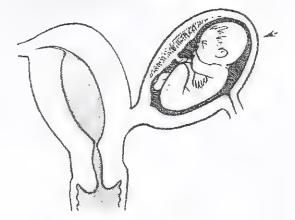
dose is repeated if no decline by $\geq 15\%$ bet days 4...7

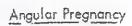
. surgery is done if no response after 3 doses

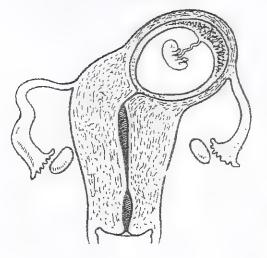
- ... why: Arios Etella reaction- to reduce with drangel D₂C may be done..... bleding.
- If $Rh ve \rightarrow give anti-D$

day 1, 4, 7.

Pregnancy in Rudimentary Horn

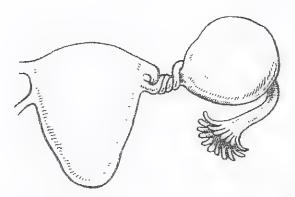






		Abortion	Ectopic
Symptoms	Triad - Amenorrhea - Pain - Bleeding	Am → bl → pain Usually present Colicky ± backache Bright red	Am → pain → bl Short period Colic, dull, sharp Slight dark
Examination	- Shock - Abdominal - Uterus - Adenexae	Proportional to bl Mild pain = period of amen No swelling	Not proportional T, R, RT Usually < 8 weeks Swelling + tender
	NI.		

imp



TORSION of PEDICLE of OVARIAN CYST.

Anastanotic area between

.....Rare types of ectopic ()

☆ Ovarian pregnancy

• Usually 2^{ry} to tubal pregnancy

• 1 ry is diagnosed by Spiegelberg criteria

Tube on affected side is healthy

- Gestational sac occupies position of the ovary

- Gestational sac is connected to uterus by ovarian ligament

- Ovarian tissue is found in the wall of the sac

☆ Pregnancy in rudimentary horn

- Usually presents late at 16 20 weeks
- It is medial to the round ligament while tubal pregnancy is lateral
- Treatment → remove horn

☆ Angular (cornual) pregnancy

• At uterine orifice of the tube, late diagnosis (14-16), more bleeding

• If disturbed → - wedge resection & repair of part of the uterus
- May need hysterectomy - Completed Family

☆ Cervical pregnancy ⇒ ttt:

• Hysterectomy if severe uncontrolled bleeding

• Conservative measures: Dand C is CI serve bleeding:

- Suturing at 3, 9 o'clock

- Silk suture around the whole cervix (as in cerclage)

- Balloon tamponade (30 ml) by Foley catheter

- Bilateral uterine artery embolization by gel-foam

2] Methotrexate local injection in the sac

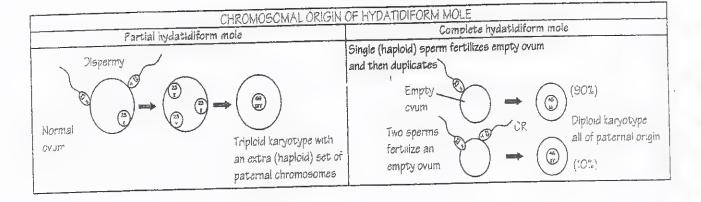
......DD of ectopic

1] Pain from

- Acute salpingitis → no amenorrhea, no fainting, fever, pain (usually bilateral), leucocytosis
- O Complicated ovarian mass or fibroid
- Acute appendicitis → no amenorrhea, vomiting, pain usually periumbilical then at Mcburney's point
- Acute pyelonephritis → loin pain radiating to the groins with fever & urinary symptoms
- 2] <u>Bleeding</u> from abortion & vesicular mole

.....Later on, after ectopic - -

- ς Contraception \rightarrow avoid IUCD and POP
- ¬ Prognosis → 15%: recurrence ¬, 30% infertility ¬



* Vesicular (Hydatidiform) Mole **

Gestational trophoblastic disease (GTD)				
Benign 🧗 🦠		Malignant		
Vesicular mole	Metastatic	Non-metastatic	aparalamanda-mentenandakan-pelaba-terpilahan dengantan personangan penanangan pelabahangan dengahangan pelabahan pel	
ridatidifarma mala)	Oleveire	T . T	-	

(hydatidiform mole) Choriocarcinoma Invasive mole

.Placental site trophoblastic tumor

Definition

Benign tumor of trophoblast ccc by trophoblastic proliferation

+ hydropic degeneration of chorionic villi

Incidence = commonest in far east * 1/1000

Etiology unknown, m.b.d.t.

o A primary oocyte abnormality !?

o IMMUNOLOGICAL, GENETIC, NUTRITIONAL (Vit A) factors

o Risk factors → previous VM, extremes of age (>35...<20)

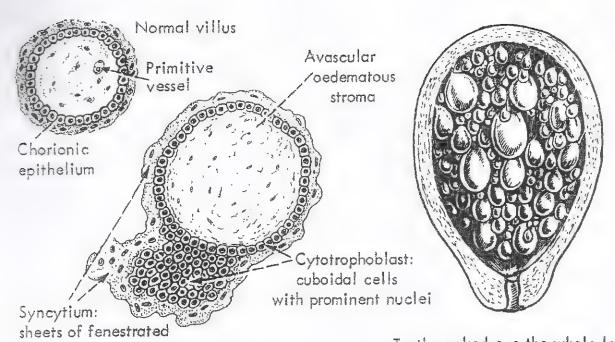
Types

1. According to pathology

	Complete mole	Partial mole
Incidence	More common	Rare
Malig. change	5–10 %	Rare
Karyotype [¤]	46 xx ✓ (all paternal)	Triploid e.g. 69 xxy ✓
Pathology	Only vesicles	Vesicles + fetus → usually
	(no fetus)	aborts in midtrimester
Etiology	Fertilization of one ovum	A normal ovum fertilized
(Androgenesis ⁿ)	by [2 sperms or rarely 1 sperm that divides into 2] followed by \(\gamma\) disappearance of all maternal chromosomes	by 2 sperms or 1 sperm with 46 chromosomes (unreduced genome)

2. According to behavior

- > Benign
- ► Invasive mole 15% (choriadenoma destruens) → if perforating the uterus i.e. locally malignant (rarely metastasize)
- ightharpoonup Metastasizes to lungs $^{\pi}$) ightharpoonup resolves with ttt



cytoplasm containing

To the naked eye the whole looks like a bunch of grapes.

typesot V. or according to behaviour:

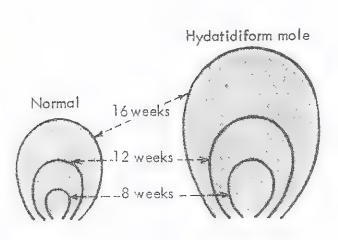
1- Berign V. or.

2- b Cally resure V.M. & Be nym

2- refarting it in V. M.

3- refarting V. M.

43- refarting V. M.



Pathology

Macroscopic

Lordifteent stages ① Uterus → enlarged, studded by vesicles (2 mm to 2 cm)in diameter, each with a small pedicle & contains semitransluscent fluid. No FETUS or PLACENTA

May be partial or complete

• May affect one twin & not the other

has LH likeaction - 1 the cacells - from control of lar manufaction o β.HCG released from the proliferating trophoblast]. They

DISAPPEAR SPONTANEOUSLY 2-3 months post-evacuation = Commpression of BUS abliteration = Avoscular pattern. Microscopic

- Trophoblastic proliferation (both cyto- & syncitio- trophoblast)
- Hydropic degeneration of C.T. stroma of villi → vesicles
- No blood vessels (<u>Avsascular</u> Pattern of Villi)

Clinical picture

♦Symptoms

- ► amenovihea + sympt. of early pregnancy + excessive abd. enlarge
- ➤ Uterine bleeding (continuous trickling) ± Veuides.
- Fain.....but no fetal movements
 - Dull aching (uterine stretch)
 - Colicky (expulsion)
 - Sharp (perforation) (due to TIUP) -

- ACUTE ABDOMEN (complicated theca lutein cyst) Rup have.

♦ Signs

- ➤ General ⇒ ill, anemic / shocked ± signs of comp.
- Abdominal
 - Uterus > period of amenorrhea
 - Uterus doughy in consistency (vesicles with no fetal parts)

- No fetal parts or FHS (except if Perthal a . Trulins.) - Bilateral enlarged ovarian swellings + ore V.M

➤ Vaginal ⇒ passage of vesicles is diagnostic (rare) Pathegrowic.

Complications $\Phi\Phi$

	AND THE RESIDENCE OF THE PARTY	
	General	Local
THCG -	1	Haemorrhage , infection
1//	⁹ Hyperemesis gravidarum	Perforation - du to Invalon (MIUP)
A Human	~Thyrotoxicosis	$Malignancy$ (choriocr.) \rightarrow %??
Thyrotropin.	- Thyrotoxicosis Pulmonary embolism & DIC	Recurrence (1-2% =) >~ WThin 1 y

Criteria of Possible development of Charlocardinano: 1-BHCG ing titre, Clather titre, the after-ve 2-I regular bleeding. 3-ty-Gitssis-metastasis.

Hysterotomy X

- * Ensures complete removal but
 - Disseminate vesicles
 - Leaves a weak scar
- * Previously performed if → severe uncontrollable hge
- * Now...it is considered a part of history of medicine

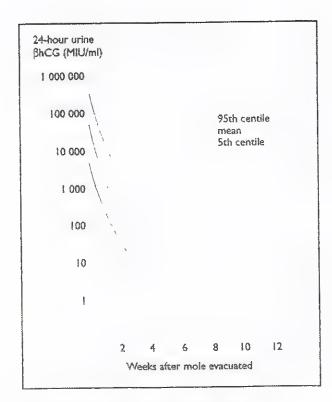
Prophylactic chemotherapy

... A single course methotrexate (or actinomycin D)
may be given at time of evacuation / hysterectomy to

- Dembolisation & metastasis
- ♣ local uterine invasion

...esp if high risk for recurrence / persistence / metastasis **

- Old age (> 40 years)
- β-hCG > 100.000 mlU/mi
- a Excessive uterine enlargement
- Theca lutein cysts > 6 cm



Hormone follow-up of benign trophoblastic disease (mean and 95% confidence limits).

nvestigations

1- Ultrasound ✓ (the best) → <u>SNOW STORM</u> appearance → Amniography → HONEY COMB appearance XX

2- β -hCG +ve in high dilutions > 100.000 (more important for <u>follow up</u>)

3- Radiography: / Plain X-ray: no fetal skeleton Chest X-ray: for metastasis

Treatment

Resuscitation +

1] Suction evacuation 🗸 by a wide bore cannula

± curettage to ensure complete evacuation (risk of perforation)

± ecbolics to ♥ hge (risk of embolism if induction is started by ecbolics)

- Don't forget →. anti-D if Rh-ve

. specimen is sent for histopathology

2] Hysterectomy (in toto)

- In old patients (> 40 years) who have completed their families to □ risk of choriocarcinoma (35% at this age)

- Hysterectomy doesn't prevent metastasis (.: follow up by β-hCG)

- Theca lutein cysts are not removed surgically ** except if complication occur & (e.g. torsion or rupture)

Follow up

> Bu B-subunit of HCG-

Sifue to -> chono carlama. Es if I then Tagain -> reccurrence

- Every week → till –ve for 3 successive times (<5 mIU /ml) - Usually becomes -ve within 2-3 months + The Car Untingst disaffer

- Every month \rightarrow for 1-2 year/s

Pregnancy is avoided for 1-2 year/s:

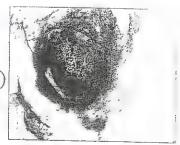
- To \$\text{\$\Pi\$ recurrence & choriocarcinoma}\$

- COC is used (IUCD x causes irregular bleeding =) ascherio Carcina Causes irregular bleeding bleeding.

bleeding of the (ITTO F V.m) is Consider al chario carcina and ill proved the Criteria of possible development of charlocarcinonia TOP

" METHOTREXATE)

- β-hCG levels are:
 - Rising (doubles in 2 weeks)
 - Plateau (failure to ♥ within 3 weeks)
 - Returning +ve after being -ve
- Persistent or recurrent uterine bleeding
- Any evidence of metastasis e.g. chest x-ray
- Biopsy → diagnostic of choriocarcioma



Chapter

Anteparium hemorrhage

Vasa Previa Placenta Previa Accidental hge

KEY POINTS

- Nonobstetric causes of antepartum hemorrhage include cervical and vaginal lacerations, hemorrhoids, infections, and neoplasms.
- ·2. Patients typically present with spotting rather than frank bleeding.
- 3. Nonobstetric causes of antepartum hemorrhage generally require simple management and have good outcomes.
- Cusco sepeculum examination of the vagina & cervix is very helpful

KEY POINTS

- 1. Fetal vessel rupture is a rare obstetric complication, usually associated with multiple gestation.
- 2. It is due primarily to velamentous cord insertion.
- 3. It is associated with a perinatal mortality of 50%.
- 4. Patients may present with vaginal bleeding and a sinusoidal FHR pattern.
- 5. Fetal vessel rupture usually requires an emergency cesarean section.

** Antepartum Haemonkage **

Definition => Bleeding from the genital tract after 20/(28) weeks till before delivery of fetus

Etiology

Placental site bleeding	Extra-placental (incidental)	Vasa previa	-The most
1. Placenta previa (inevitable hge) 2. Placental abruption (accidental hge ✓✓)	 Local gynecological cause Excessive show Marginal sinus bleeding Rupture uterus 	(the only cause of fetal hge ")	of APH.

..... - ...Vasa Previa ... 5...

The only Cause of fetal Hge.

Definition = fetal hge due to tear of umbilical vessels running between the presenting part & cervix

Incidence

→ very rare: 1 /5000 with fetal mortality: 50–75%

The statement of the state

Etiology - Velamentous insertion of the cord (defect of war tonge - Placenta succenturiata, bipartate placenta

Diagnosis

of the Sympt. → APhge (<u>mild</u> bleeding but <u>marked</u> fetal distress) → Vessels are felt as cord like hands crossing amniation

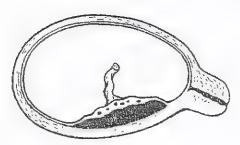
→ colored Doppler may show the vessels

Investigations

11 5	9		
/		Fetal blood	Maternal blood
	Hb%	16-18 gm%	10-12gm%
	Hb electrophoresis	Hb F	Hb A
	Blood group & Rh	May differ	May differ
	Acid ellution test المامي أوارح	RBC's not haemolysed	Haemolysed
/	(Kleihauer Betke test)	(acid resistant)	(ghost RBC's)
- /	Alkali-denaturation test	No color change →	Blood turns \rightarrow
r.F	(Apt test) 0.25% NaOH	Pink (alkali resistant)	light yellow/brown
	GC. T (Alkali-Preceptation	Ket).	

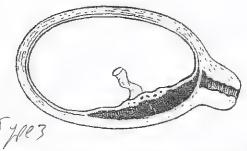
Treatment → immediate delivery usually by CS-*

→ rarely forceps or ventouse if fully dilated

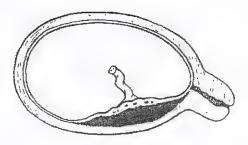


Typel

Thelower margin of the o acenta reachthe lower segment. Low

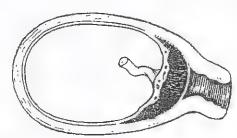


The place to but six the internal of whendo to but not when fully the internal or "incomplete").



Type 2

The placenta reaches the internal as when closed but does not cover it. ('Marginal').



Туре 4

The placenta covers the os even when the cervix is fully dilated ('Central' or 'Complete').

Placenta Previa

Definition \Rightarrow BLEEDING from within the genital tract AFTER 20 / 28 wks & BEFORE delivery of the fetus DUE TO a placenta situated in the lower uterine segment

Incidence $\Rightarrow 0.5\%$

→ More common in MULTIPARA PREVIOUS UTERINE SCAR *

→ Recurrence rate....4–8 %

Etiology /

(Sasal

theory de Seded Delayed development of chorion frondosum

- Delayed disappearance of zona pellucida

- Deficient decidua (1 parity, 1 age , endometritis) Rewins CS.

Persistence of villi in the decidua capsularis

Large placenta

- Twins, D.M., RH > due to hydrops fetalis. - Placenta membranacea Cong. anonely 15-20 cm -> 15-20

Classification dependentime of implantation; more delayed implantation ->

- I	1 deg	Wee.		
Cont of	1 st o	PP lateralis "low lying" //	60%	Lower margin of the placenta lies in LUS but not reaching the margin of internal os
40 th 1	2 nd °	PP marginalis "marginal"	30%	Lower margin of the placenta reaches the margin of the internal os
nator {	3ra o	PP centralis incomplete "partial"	7%	Placenta partially covers the internal os
10%	4 th °	PP centralis complete "total"	3%	Placenta completely covers internal os

Pathogenesis

Pregnancy:

Placenta is inelastic so bleeding occurs due to stretch of LUS (shearing mech.). Bleeding is augmented by the inability of the weak LUS to compress the torn vessels

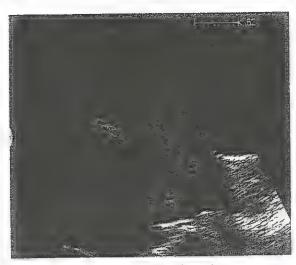
Peak incidence of bleeding is = 30=34 wk treeh. Period of whering

First bleeding episodes are usually = mild and and onet

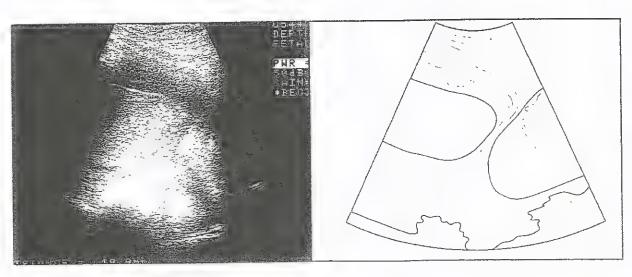
<u>Labor</u>: d.t. ex dilatation (rarely may occur for 1st time in labor)

PV is CI if there is bleeding or PROM.
PV is CI after 28 w except after exclusion of PP.





An anterior placenta praevia extending to just beyond the internal os.



Placenta praevia – type two.

Clinical picture

Symptoms --> Bleeding: as Wis verk - Fresh bright red wear vag. Inothine for clothing.), Not retained. Painless.....except if ... associated with labor pains -Causeless.....except if...after intercourse or PV - Recurrent except if ... placenta is just reaching the LUS first prosentation during labour. ⇒ Signs

♦ General → pallor or shock (according to degree of bleeding)

Abdominal

• Palpation 25 moffe lover -> Tuins, large slocute, DM

- * Fundal level → corresponds to period of amenorrhea
- * Umbilical grip → Lax uterus, not tender
- * Pelvic grips → non-engagement ± malpresentations (30%)
- Auscultation
 - * FHS are normal except in severe cases (more than 1/2 placental separation is needed for fetal distress to occur, this is more in cases of)

Vaginal

- · Contraindicated & > for fear of place to P.
- Except if 1 the patient is in labor 2 has minor degree
 - Aim is to determine the possibility of labor
 - In the operative theatre which is ready for immediate interference by CS + available blood
 - This is called a double set up technique [2 teams]
 - Placenta (if felt) will be a fleshy tough sponge

Investigations (ultrasound)

- * The only & best method used (98% sensitivity)
- * Repeated serially (every 2 wks) to detect upward migration: The apparent upward movement of the placenta from the LUS (due to unequal growth of UUS & LUS). This may lead to disappearance of p.previa or lessening of its degree
- * Thus:-
 - P. previa is *more* common at earlier gestational age
 - P. previa is more likely to persist if diagnosed after 30 wks
- * Other methods:- 🖣 Angiography
 - MRI → very accurate but expensive
 - Thermography → more temp. over placenta

Risk factors for placenta accreta

- Previous retained placenta
- High parity
- Advanced maternal age
- Placenta praevia
- Previous caesarean section
- History of dilatation and curettage or suction termination of pregnancy
 Previous postpartum endometritis

Classification of abnormal placental attachment

Glassification of astronomy				
Туре	Incidence	Pathology		
Placenta accreta	75–78%	Invades superficially into the myometrium		
Placenta increta	17%	Invades deeply into the myometrium		
Placenta percreta	5–7%	Invades through the myometrium and penetrates the outer serosal layer of the uterus. It may invade adjacent structures, including bladder and bowel		

ΦФ

Complications

→ Maternal

- ◆ Pregnancy → APhge (anemia if mild, shock if severe)

1st stage

- Prolonged labor (due to hge & uterine inertia)
- PROM (presenting part not well fitted on the cervix) cord prolapse & infection

2nd stage: difficult (obstructed labor + malpresentations)

3rd stage: PPHge

- Atonic (poor contraction of LUS + poor maternal condition)
- *Traumatic* → friable lower uterine segment.
- Retained placenta (5%) → placenta accreta (d.t. poor decidual development) → incidence ↑ with no of CS *

.....(APhge predispose to PPhge)......(APhge weakens, PPhge kills)......

♦ Puerperium ...5³

Sepsis

- Poor general condition (shock + exhaustion)
- Placenta (friable + near vagina + retained parts)
- Premature rupture of membranes
- Increased surgical interference

Secondary postpartum hge (retained placental parts).

Subinvolution of uterus

Fetal

♦ Preterm laborPTL	} due to
♦ Intrauterine growth retardation IUGR	} poor
♦ Congenital fetal malformationCFMF) blood
♦ Intrauterine fetal deathIUFD	supply

Maternal mortality (<1%) Perinatal mortality (5%) - Hemorrhage - All complications Sepsis - Especially → { Prematurity }

A Care of the newborn

1] Conservative ✓✓ if → . Bleeding → mild . Fetus \rightarrow not mature, not distressed . Mother \rightarrow not in labor ☆ Hospitalization...aim: keep condition under control till maturity 1. Mother: - Bed rest, no P/V, no vaginal douching Correct anemia by diet, iron ± blood transfusion. - Close observation, ready blood stores 2.Fetus: - Give steroids for lung maturity - Serial tests for fetal well being 3.Placenta: follow up placental migration by U/S 2] Termination if → Bleeding → severe......or . Fetus → mature or distressedor . Mother \rightarrow spontaneous onset of labor ☆ Anti-shock measures ✓✓ & Birth either # C.section 1: 3- + severe bleeding, 2nd posterior, 3rd, 4th ► LSCS → . better control of bleeding (near placental bed) . leaves a strong scar If placenta is found anterior - Incise the placenta & deliver baby through it ✓ - Reach round it till head is felt Control of placental bed hemorrhage - Ecbolics + massage + hot packs - Under-running sutures - Uterine artery ± bilateral internal artery ligation - Supra-vaginal hysterectomy # Vaginal: 3- + mild bleeding, 1st & 2nd anterior (why?) ⇒ AROM ± oxytocin \circ I^{st} stage \rightarrow continuous fetal & maternal monitoring o 2^{nd} stage \rightarrow no forceps or breech extraction 3rd stage → guard against postpartum hge ☆ Care of the maternal complications......shock, PThge

Abruptio placenta (accidental hge)

Definition BLEEDING from the genital tract of placental site origin

AFTER 20 / 28 wks & BEFORE delivery of the fetus

Due to separation of a normally situated placenta

Incidence = 1/200-500 [> in PG with PIH, elderly PG, GMP]

Eticlogy Φ idiopathic in many cases.....

1] PDF: . ^ age, parity....smoking, alcohol

.↓ folic acid *, vit.C., vit.K

. Previous accidental hge

21 Maternal disease:

- Preeclampsia (the most important → 50%) ✓✓
- Vascular wall defect (DM., collagen disease)

3) Trauma to the abdomen

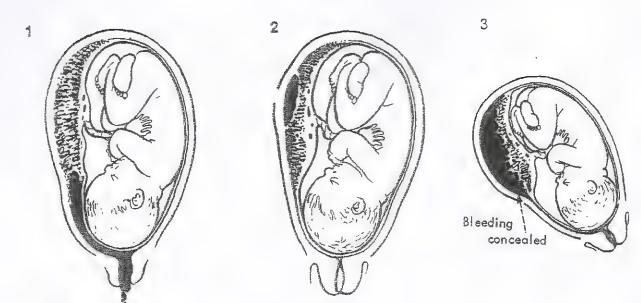
- Accident.
- External Cephalic Version (esp if traction on short cord)
- 4] Sudden 🕂 in intrauterine pressure:
 - After sudden ROM in polyhdraminos
 - After delivery of first twin.

5] Placental abnormalities :

- Circumvallate placenta
- Placenta implanted on scar, septum, fibroid

Pathogenesis

- Injury of vessels in the choriodecidual space → retroplacental hematoma → <u>automatic</u> extension due to rupture of more vessels by the collecting hematoma → more expansion of the hematoma
- Blood may escape between muscle fibres of myometrium (<u>Couvelaire</u> uterus / uteroplacental apoplexy) → black flabby uterus incapable to contract
- Extension of blood may produce ecchymoses below peritoneum or even <u>rupture</u> uterus & internal hemorrhage
- Release of tissue <u>thromboplastin</u> + <u>consumption</u> of the clotting factors within the hematoma → will lead to DIC → FDP's:- (renal failure + tocolytic effect → atonic PPHge)



External bleeding alone causes little upset.

	s grades of accidental haemorrhage Severity of bleeding		
	Mild	Moderate	Severe
Pulse	No change	Raised	Raised
Blood pressure	No change	Lowered	Lowered
Shock	None	Often	Always
Oliguria	Rare	Occasionally	Common
Hypofibrinogenaemia	Rare	Occasionally	Common
Uterus	Normal	Tender	Tender and tense
Fetus	Alive	Usually dead	Dead
Blood loss (litres)	< 1	1-3	3-6

Types

1] According to bleeding

_	
1. Concealed	The blood separates part of the placenta but does
(10%)	not reach the vagina
2. Revealed	The lower margin of the placenta separates and the
(30%)	blood tracks down wards (between the membranes
	& uterus) to escape through the vagina.
3. Mixed (60%) 🗸	Commonly starts concealed then become revealed

2] According to severity

		C/P	Fetus	Shock	DIC
Class 0	Mildest	-ve	Alive	-ve	-ve
Class I	Mild	+ve	Alive	-ve	-ve
Class II	Moderate	++ve	Distressed	+ve	-ve
Class III	Severe	+++ve	Dead	++ve	+ve

Clinical picture

1] Symptoms

- Pain ✓ (SUDDEN, SEVERE, CONTINUOUS abdominal pain)
- Bleeding....dark, clotted (absent in concealed type)
- Shock (hypovloemic + neurogenic in concealed)

2] Signs

> General

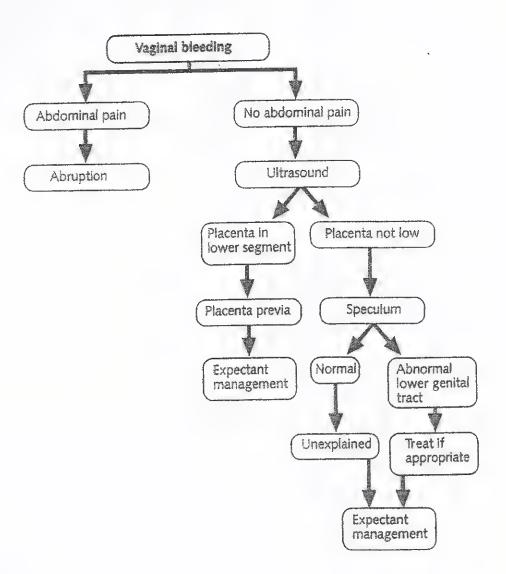
- Signs of etiology e.g. PIH (but arterial blood pressure may be apparently normal i.e. hypotension due to shock is masked by PIH (decapitated B.pr.) : Hypovolemia is better detected by CVP
- Shock [may not correspond to the external bleeding]
- Signs of complications e.g. DIC

> Abdominal

- Palpation:
 - * Fundal level → higher than period of amenorrhea
 - * Umbilical grip \rightarrow \uparrow basal uterine tone (board like rigidity)
 - * Pelvic grip -> normal presentation + engaged head
- Auscultation: according to severity (distressed ✓ or absent)

> Vaginal

- Contraindicated (No PV in any case of APHge)
- Only done after exclusion of P.previa by U/S → well engaged head & very tense membranes (if ruptured → bloody liquor)



Investigations

- 1. $Etiology \rightarrow preeclampsia$
- 2. Diagnosis (U/S) → . exclude placenta previa ✓ . may find retroplacental hematoma
- 3. $Complication \rightarrow DIC$, renal function tests

Differential diagnosis

- 1. Causes of acute abdomen in late pregnancy (concealed or mixed)
- 2. Causes of antepartum haemorrhage (revealed or mixed)

	Mixed accidental hge.	Placenta Previa
History	- Once	- recurrent
- bleeding	: - has etiology	- causeless
है । ।ि. व यक्ता	- painful	- painless
	i - dark clots	- fresh blood
Examination		
- General	* Etiology e.g. → PIH	* no etiology
	* Shock > hge.	* shock = hge.
	* DIC	
- Abdominal	Tender, hard	Painless, soft
- Vaginal	- no placenta	- placenta felt
	- cephalic	- malpresentation
	- well engaged	- not engaged
<u>2\µ</u> (normally situated	in LUS

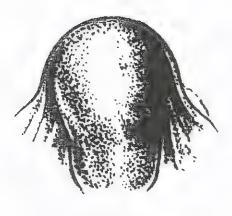
Complications

中盆

- ⇒ Maternal [MMR..... 1%] "
 - Due to the developing hematoma
 - 1] Shock
 - . Due to......APhge, PPhge (atonic $\checkmark \pm$ traumatic \pm DIC)
 - Leading to Renal failure (shock + PET + DIC)
 - Sheehan syndrome
 - 2] DIC \rightarrow accidental hge is the commonest cause of DIC
 - Due to the high intrauterine pressure
 - 3]Couvelaire uterus (utero-placental apoplexy) & Rupture uterus → intra-peritoneal haemorrhage
 - 4 Amniotic fluid embolism
 - <u>♠ Due to the etiology</u> e.g. complications of preeclampsia
- ⇒ FETAL: PTL ✓..... IUFD ✓..... IUGR.....(why?)

History

Alexandre Couvelaire (1873–1948) was the first to describe extensive haemorrhage into the myometrium; he recognized that it was impairing the myometrium's ability to contract, such that in the case he reported, a caesarean hysterectomy was required. He also was an early proponent of caesarean section for placenta praevia.



Couvelaire uterus

KEY POINTS

- Placental abruption accounts for 30% of all thirdtrimester hemorrhages.
- 2. Fetal mortality rate can be as high as 35%.
- Patients usually present with vaginal bleeding, painful contractions, and a firm, tender uterus;
 20% of cases present with no bleeding (concealed hemorrhages).
- 4. Major risk factors include hypertension (chronic or gestational) and previous history of abruption.
- Placental abruption can be complicated by hypovolemic shock, DIC, and preterm delivery.
- Patients can be delivered vaginally if they are stable; cesarean delivery is necessary in the unstable patient or when fetal testing is nonreassuring.
- 7. Risk of recurrence increases in subsequent pregnancies.

Treatment

1] Termination vis the usual fate, as:-

- \circ Bleeding is usually \rightarrow severe
- o Fetus is usually \rightarrow distressed
- Mother usually enters in → spontaneous labor
 - a] Anti-shock measures
 - b] Birth by:

1- Cesarean section ✓✓: ⊶+

- Maternal or fetal distress & delivery is not expected soon
- MOST IMPORTANT → coagulation defects should be corrected first
- Hysterectomy → in severe atony or Couvelaire * (if failed to contract)

2- Vaginal if: 🖘 +

- Especially if the fetus is dead or the patient is advanced in labor
- Usually easy (well engaged head) & rapid (\(\shcape{e}^{ed} \) basal uterine tone)
- Early AROM (why......3R) ± oxytocin
 - Relieve.....the high IUPr
 - Reveal.....any internal hge.
 - Release.....PG...accelerate labor

1stage → continuous monitoring (F & M)

2nd stage → usually rapid

3^{td} stage → guard against PPhge

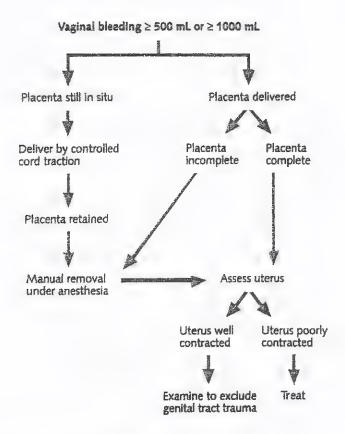
- c] Treatment of etiology (PET) & complications (DIC)
- d Care of the newborn

2] Conservative, rare

- Very rare (chronic abruption): as once abruption occurs, there will
 be → automatic extension → severity → complications
- Indications:
 - 1. Bleeding → mild (usually revealed or small retroplacental hematoma).
 - 2. Fetus \rightarrow not mature < 37 wks
 - 3. Mother \rightarrow not in labor
- \circ Aim to \rightarrow control the condition till fetal maturity.
 - Mother:
 - Fetus:
 - Placenta: serial U/S to follow size of <u>hematoma</u>
- o Continue → till any of the indications of termination occur

Redpenium Hemonice

Atonic
Traumatic
Retained Placenta
DIC
Acute Inversion



Algorithm for the management of early PPH.

Causes of PPH

- Tone: uterine atony
- Tissue: retained products of conception
- Trauma: genital tract laceration
- Thrombin: clotting abnormalities

Postpartum Haemorrhage

Definition

⇒ It is haemorrhage from the genital tract

AFTER delivery of the fetus

TILL the end of puerperium **EITHER** ?

- To a degree affecting maternal general condition
- More than 500 cc
- Causing haematocrit drop > 10 %
- ▶ Incidence has been reduced from $15 \rightarrow 5\%$ (d.t. ↑ use of ecbolics)
- ▶ It is the commonest cause of MMR in developing countries (30%)
- ➤ More common with history of previous PPHge *

Types

- Primary PPhge (hge within 24 hours of delivery)

 - Trauma......Troumotic......(extra-placental site hge)

 - Thrombosis...Coaquiation defects......(mostly DIC)
 - Acute inversion of the uterus.....(v.v.rare)
- \Rightarrow Secondary PPhge (hge after 1st day till end of puerp.) Φ Φ
 - 1- Etiology
 - a- The commonest.....retained fragments of placenta ± infection
 - b- The most serious.....choriocarcinoma
 - c- Sepsis..... separation of a slough → bleeding
 - d- Subinvolution.....inversion of the uterus
 - e- Submucous polyp....if ulcerated
 - f- Others
 - **Lical** gynecological disease → cervical ulcer
 - **Empra** → coagulation defect

2- Assessment

- * History (mode of delivery)
- * Examination (general, abd, local: cusco)
- * Investigation (U/S, β-HCG)

3- ttt of the cause

- * Retained fragments......
 - if small remnant / mild bleeding → methergine + antibiotics
 - if large remnants / severe bleeding → evacuation guided by U/S
- * Choriocarcinoma...... chemotherapy

1 Atonic PPhge

Definition

- * Postpartum bleeding due to weak contractility & retractivity
- * Constitutes about \rightarrow 90% of cases (the commonest $^{\pi}$)

Etiology D

[i] During prognancy

- APhge (pl. previa, accidental hge)
- Maternal disease (anemia , pre-eclampsia)
- Over-distended uterus (twins , polyhydramnios)
- Long use of tocolytics

[2] During labor

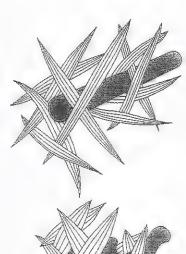
- ⇔ 1st Stage → Prolonged 1st stage
 - Excessive straining
 - Overuse of sedatives
 - Chorioamnionitis
 - Full bladder / rectum
- ⇒ 2nd Stage → Prolonged 2nd stage
 - Excessive manipulation
 - Deep anesth. esp. halothane
 - Precipitate labor
- ⇒ 3^{ra} Stage ⇒ Retained parts of placenta

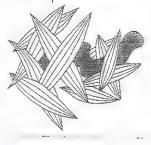
[3] Causes in the uterus

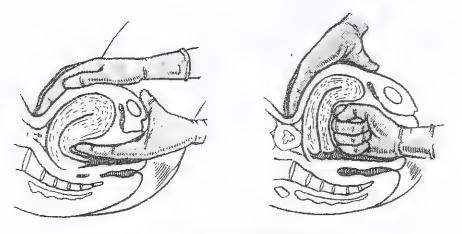
- Multiple fibroids
- Congenital malformations
- Grandmultipara

Clinical Picture

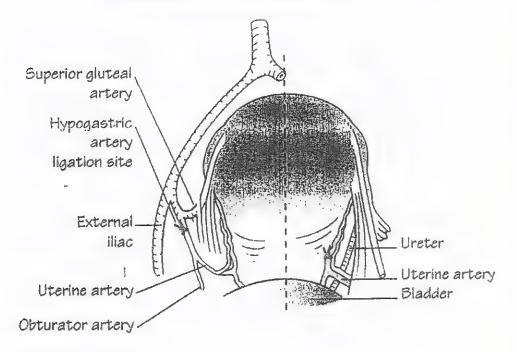
- > History -> severe vaginal bleeding after delivery of the fetus & placenta
- > Examination
 - $General \rightarrow Shock$
 - Abdominal → Uterus soft & enlarged
 - Vaginal → to exclude traumatic PPhge.

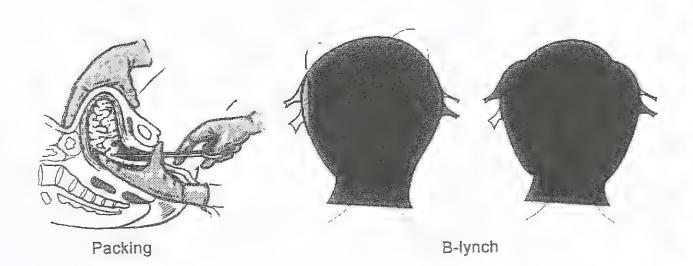






Bimanual Compression





Treatment

☆ Prophylactic

- Avoid all pdf. → proper ANC
- Proper management of 1st, 2nd & 3rd stages of labor

☆ Active: Resuscitation +

1st line - massage "

- Ecbolics (oxytocin, methergine, PGE₁ misoprostone 800µg –)
- Empty bladder + stop halothane

2^{nd} line \Rightarrow exploration of birth tract under G.A:

- Exclude trauma
- Evacuate blood clots or retained placental parts

3rd line Dimanual compression of the uterus

- Closed fist of the right hand is placed into anterior fornix
- Left hand is placed abdominally to compress the uterus inbetween → kink uterine vessels & compress placental site
- This is continued with the aid of assistant every 15 min.

4th line = laparotomy

- ➤ Pt. completed her family → Supravaginal hysterectomy
- ➤ Pt. not completed her family →
 - Direct uterine massage, hot fomentations
 - Intra-myometrial prostaglandins " (PG-F_{2α})
 - Bilateral uterine & ovarian artery ligation
 - Bilateral internal iliac a. ligation
 - Difficult surgical technique, with possible injury to ureter, IIV
 - Uterine blood supply will then depend on collaterals
 - If all failed → Supravaginal hysterectomy

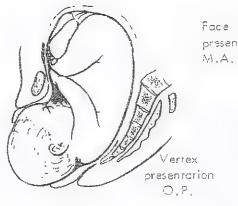
<u>Recently</u> → . B-lynch operation (Brace suture)

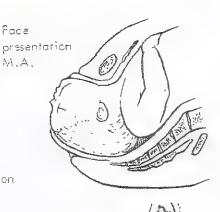
- . Balloon (hydrostatic intrauterine temponade)
- . Bilateral uterine a. embolization using polyvinyl-alcohol particles (gelfoam)

<u>Previously</u> → methods not done now xx

- * Uterine douche by Bozeman double way catheter using warm saline or antiseptic → stimulate contraction, remove remnants.
- * Uterovaginal pack for 24 hrs (Abcs + ecbolic + catheter) → stimulate contraction, pressure over bleeding site.

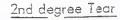






ist degree Perineat Tear

Vaginal and perineal skin are torn, but the cerineal muscles are intact.



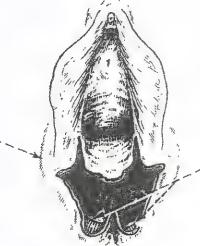
The perineal body is torn right down to (and sometimes partly involving) the anal sphincter. The vaginatears often extend up both sides of the vagina.



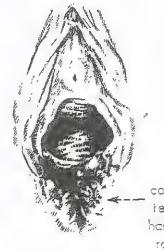
3rd dagree Fear - "Complete Tear"

The whole anal sphincter is torn apart, and there may be a tear of the rectal wall. Note how the enas of the sphincter muscles tend to retract.

This injury, if not repaired, leaves the patient with faecal incontinence.



Torn ends of anal sphincter



A complete tear that has failed to neal

2 Traumatic PPhge

[1] Perineal tears

€tiology: Φ

1] Over stretch of perineum

- If head is allowed to extend before crowning
- Malpresentation: Face, DOP
- Large head
- Narrow vaginal introitus or subpubic arch

2] Papid stretch of perineum

- Precipitate labor
- After-coming head of breech.

3] Causes in perireum

- Rigidity (e.g. elderly PG or previous scar)
- Edema (e.g. PET or obstructed labor)
- 4] <u>Injury of perineum</u> \Rightarrow forceps...ventouse....destructive operations

Degrees (incomplete or complete)

1st degree -> vaginal wall + perineal skin

 2^{nd} degree \rightarrow + perineal muscles \pm levator ani

 3^{rd} degree \rightarrow + external anal sphincter

4th degree → + rectal mucosa (some consider it 3^{td} degree)

* Hidden perineal tear

Tear in the perineal muscle without any visible tear of the vagina or skin \rightarrow predisposes later on to rectocele

Complications

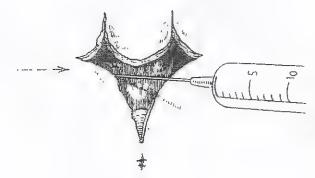
 $\underline{Early} \rightarrow \text{Hge} \pm \text{infection}$

<u>Late</u> . incomplete tear $(1+2) \rightarrow \text{prolapse}$

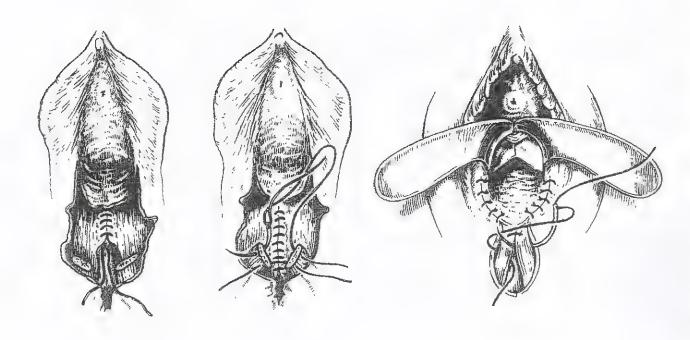
- . complete tear $(3+4) \rightarrow$ incontinence of flatus & stools
- . improper healing \rightarrow recto-vaginal fistula
- poor healing → scar → dyspareunia

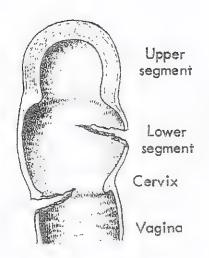
Treatment

- → Prophylaxis = proper management of the 2nd stage ± episiotomy (if there is overstretch or the perineum is about to tear)
- → Active = immediate surgical repair (within 24–48 hrs of delivery)
- → Old complete perineal tear
 - If later → wait 3-6 m (tissues may be edematous ± infected)
 - Anatomical repair in layers [Lawson Tait operation]



Local
infiltration
anesth.
lignocaine 1%





Technique of repair

- * Local infiltration is better than GEA
- * Interrupted sutures are better than continuous
- * Vicryl is better than chromic catgut
- * Sutures are taken from above downwards
- Rectal mucosa → INVERTED LAMBERT sutures (to avoid mucosa)
- Ext. anal sphincter → approximate the 2 dimples at sides of anus (torn ends)
- Deep perineal muscles ± Levator ani
- Vaginal wall → continuous or interrupted stitches
- Superficial perineal muscles & then → perineal skin closure

Post-operative care

- Minor degrees → local cleanliness
- Major degrees →
 - perineum: dry, clean, antiseptic as betadine (povidone iodine)
 - diet: NPO for 48 hrs, then → low residue + increased fluids
 Laxatives are used for 2 weeks (stools should be soft)
 - systemic antibiotics......intest. antiseptics (neomycin + flagyl ..5ds)
 - no rectal suppositories.......No sexual intercourse for 2-3 ms

[2] Vaginal tears &

Etiology: as perineal tears

Diagnosis

- Traumatic PPhge (fresh blood +contracted uterus)
- EUA (with good light + retraction by Sim's speculum
 + Auvard self retaining post vag wall retractor)

Complications

 $Early
ightharpoonup Hge (sometimes difficult to control <math>\checkmark$) \pm infection COLPORRHEXIS (rupture of the vaginal vault or post. fornix)

<u>Late</u>

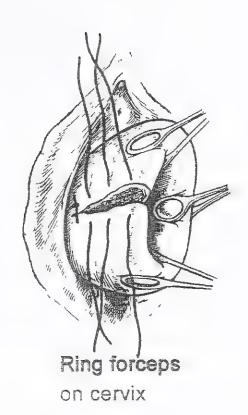
- If bladder is involved → vesico-vaginal fistula or incontinence
- If rectum or sphincter → recto-vaginal fistula or incontinence
- If levator ani → prolapse
- Poor healing → vaginal stenosis → dyspareunia

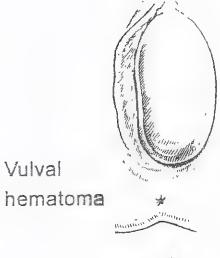
Treatment \Rightarrow resuscitation 1st

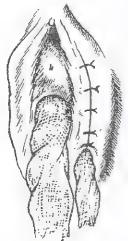
- Immediate repair (from above downwards)
- If failed → vaginal pack + catheter + abcs (for 24 hrs)
- If failed → bilateral internal iliac artery ligation.

Sandl's ring Thinned out lower segment

Cervix tears away from vagina here







[3] Cervical tears

Etiology:

- o <u>causes in the passage</u> → cervical fibrosis
- o <u>causes in the passenger</u> → large baby
- o <u>causes in power</u> → ppt labor
- \circ <u>obstetric operations</u> \to forceps, ventouse, manual dilatation of ex

Tupes

- 1. Unilateral
- 2. Bilateral
- 3. Stellate (multiple radiating)

Diagnosed by EUA = the cx is grasped by "4" ring forceps at its 4 corners

Complications

- > Early Hge + infection
 - Rupture uterus → if it extended upwards ^x
 - Ureteric injury → during surgical repair
- ► Lata . Patulous internal os → habitual abortion & preterm labor
 - Chronic cervicitis → infertility or cervical dystocia
 - . Ectropion → eversion of cervical lips in bilateral tears

Treatment resuscitation + suturing cx tears from apex downwards NB: she may need cerclage in next pregnancy

[4] Genital tract hematomas 🔑

Etiology - Traumatic vaginal delivery e.g. forceps

. Sometimes occur in normal spontaneous labor *

☆ Vulval (infra-levator)

- Presence below levator ani → prevents its upward extension
- There is tense tender bluish fluctuant swelling at the vulva
- ttt → observation if small & localized

☆ Paravaginal (supra-levator)

- Sometimes not easily seen (felt by P/V)
- May be suspected by sense of rectal straining (due to pressure)
- ttt → evacuation only if large * + drain + packing the vagina

A Broad ligament (Sub-peritoneal hematoma)

- Progressively expanding → broad ligamentary swelling
- It may dissect its way upwards → may even reach up to diaphragm
- ttt → laparotomy: evacuation ± bilateral uterine artery ligation

Scar rupture (bleeding is less as scar is fibrotic) *

USCS (2 – 4%) ^H	LSCS (0.2 – 0.4%)
1- Thick muscle layer →	1- easy to coapt edges
difficult to coapt edges	
2- Haemostasis difficult →	2- haemostasis more easy
Hematoma weakens scar	
3- infection more common	3- infection less common
4- Muscle is active in puerperium	4- muscle less active
→ pulls on scar	
5- Placenta may implant on scar	5- less liable
in next pregnancy	ee aaraa

incidence

- Varies according to level of obstetric care $^{\text{m}}$ (1/1.000 \rightarrow 1/4.000)
- Rupture uterus is the worst complication facing the obstetrician
- It should be suspected in any patient with collapse during or after labor
- More common in MG * (96%) due to
 - Passage ⇒ weak uterine wall & pendulous abd. (→↑malpresentations)
 - Passenger ⇒↑ fetal size (&↑ % of DM)
 - Power ⇒↑↑ uterine contractions in response to obstruction 🖔
 - Attendant false sense of security

Etiology $\Phi\Phi$

→ During prognancy.....APHgo

A-Spontaneous

- Ruptured previous uterine ✓ scar * (UUS >LUS)
- Rupture of anterior sacculation \rightarrow in fixed RVF
- Rupture of posterior sacculation → in ventrofixation
- Rupture of pregnancy in rudimentary horn
- Invasive trophoblastic disease
- Placenta percreta
- Concealed accidental haemorrhage

B- Traumatic

- Trauma to the abdomen (e.g. penetrating wounds, seat belts)
- External cephalic version (ECV)

→ During labor......PPHgo

A-Spontaneous

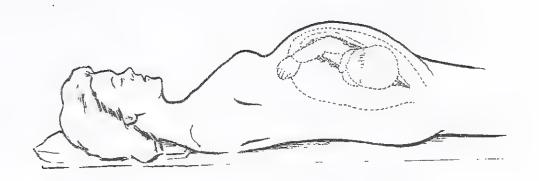
- Same etiology as during pregnancy (scar✓)
- Obstructed labor ✓✓ (the commonest)
- Maluse of ecbolics

B- Traumatic

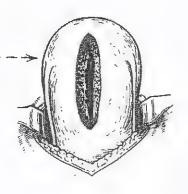
- Obstetric operations < full cx dilatation (✓forceps)
- Excessive fundal pressure
- Manual dilatation of cx or extension of a cx tear
- Manual removal of placenta "

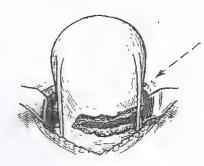
Types

- ⇒ Incomplete rupture (LUS) muscle layer is only ruptured with intact peritoneum subperitoneal hematoma (occult rupture) or









RUPTURE of a LOWER SEGMENT SCAR

SPONTANEOUS RUPTURE --



Clinical Picture

Rupture during pregnancy

1) <u>Impending (threatened) rupture of scar</u>

- * Separation of the fibrosed edges of a scar
 - . min. symptoms (tender scar) or signs (vag. spotting) d.t. fibrosis "
 - . The patient may even come to hospital walking (silent rupture)
- * U/S → gapping (dehiscence) of the scar

2) Frank rupture uterus (acute abdomen)

- Symptoms
 - Sudden severe abdominal pain, followed by
 - Collapse (internal hge: usually severe esp. if ut. vessels are torn)
- o Signs
 - General → Shock
 - Abdominal → . T, R, RT (Late: Cullen's sign, shifting dullness)
 - . Fetus felt abdominally with -ve FHS
 - . Uterus retracted away & becomes lax "
 - Vaginal →
 - . External bleeding may be present
 - . Hematuria " may be present (injury of urinary bladder)

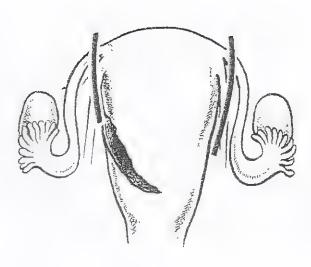
※ Rupture during labor ※

1) Obstructed labor

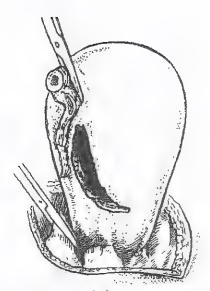
- o Symptoms: Of obstructed labor, then
 - Cessation of labor pain → sudden severe abdominal pain
 - Vaginal bleeding (& feeling of something giving way ")
 - Collapse (d.t. both vaginal & intraperitoneal hge.)
 - Cessation of fetal movements
- o Signs
 - General → shock + dehydration
 - Abdominal → as in preg.....
 - Vaginal → of obstructed labor . vulva : edematous
 - . vagina : dry hot edematous
 - , cervix : edematous or tear
 - → Plus . vaginal bleeding
 - . presenting part may recede upwards

2) <u>Traumatic rupture</u> (forceps)

- o Suspected by \rightarrow PPhge following obstetric operation
- o Discovered by → Routine fundo-perineal examination



Division of the fallopian tubes and broad ligaments, leaving behind the ovaries and part of the tubes.



After incision of the peritoneum at the site of rupture the bladder is stripped from the uterine wall and a subtotal hysterectomy performed.

Differential diagnosis

- 1] Bleeding according to time (APhge, IPhge, PPhge)
- 2] Acute abdomen in pregnancy or labor.....

Complications

1) Maternal

- * Mortality (10%) > hypovolemic shock ± acute renal failure
- * Morbidity
 - Infertility → if hysterectomy was done
 - Rupture uterus in next pregnancy → if repair was done
 - Ureteric injury during repair (esp. on left side)
 (as rupture is > on Lt side d.t. dextroratation)

2] Fetal

- * complete rupture \rightarrow 100% mortality
- * incomplete rupture \rightarrow 60% mortality

Treatment

Prophylaxis

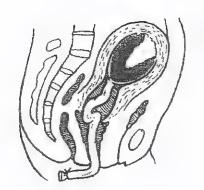
- 1-Proper antenatal care
 - * Early detection of any abnormality needing CS (macrosomia, CPD)
 - * GMP must deliver in HOSPITAL (why?)
 - * Patient with previous uterine operations must deliver in HOSPITAL
 - One LSCS → may try vaginal delivery
 - Two or more LSCS \rightarrow elective C.S. at completed 37 wks (38)
 - One USCS or hysterotomy \rightarrow always C.S.
 - Previous repair of rupture should be hospitalized all-through

2-Proper intranatal care

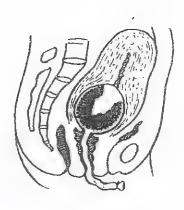
- * Early detection of signs of obstructed labor
- * Proper use of ecbolics
- * Adequate precautions in operative obstetric deliveries
- * EUA if PPhge occurred for early diagnosis

> Active

- Resuscitation
- Laparotomy 'midline incision'
 - Supravaginal hysterectomy (ideal ttt) //-LEAVE THE OVARIES-**
 - Bilateral IIA ligation may be needed to control hge.
 - Exploration of injury of other structures (bladder, ureter)
- Conservation 🗣 (repair) of uterus may be done in limited cases →
 - PG, young patient......Clear cut edges, small wound
 - Patient must be hospitalized next pregnancy



RETAINED PLACENTA



8 Retained placenta



Definition (0.5 - 1%)

Failure of delivery of the placenta within ½ hour of delivery of fetus

Physiology of placental separation

Depends on uterine contraction & retraction. The placenta being inelastic → can't cope with the ↓ in uterine length:-

- Separation of placenta at the line of cleavage between it & the uterus
- Pollowed by placental descent through the genital tract

Etiology ΦΦ

Retained separated

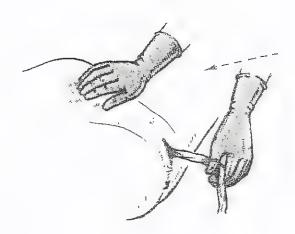
= failure of placental descent

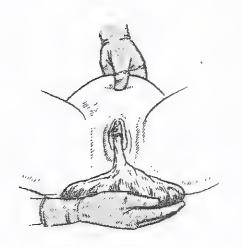
- uterine atony
- contraction (constriction) ring
- complete rupture uterus → escape of placenta to abdomen
- full urinary bladder

⇒ Retained adherent

= failure of placental separation

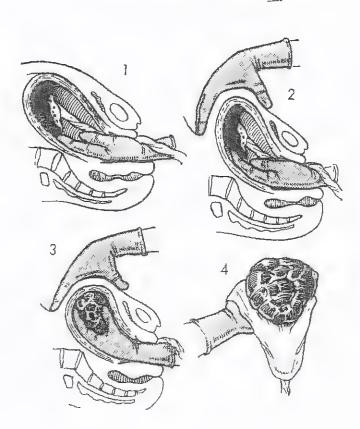
- * Simple adherence due to (uterine atony, DM, RH, syphilis)
- * Morbid adherence
 - According to degree of invasion
 - Placenta accreta vera: $<\frac{1}{2}$ myometrial invasion
 - placenta increta: $> \frac{1}{2}$ myometrial invasion
 - placenta percreta: invading peritonium even into bladder
 - According to number of cotyledons involved \rightarrow
 - adhesion may be focal, partial, total
 - Causes of adhesions (more in GMP)
 - 1. Placenta previa
 - 2. Scar tissue due to
 - Previous C.S., myomectomy
 - Previous manual separation of the placenta
 - Endometritis
 - 3. Presence of congenital uterine anomaly (as septum)
 - 4. Submucous fibroid
 - 5. Congenital absence of layer of Nitabüch





Complications of manual removal of the placenta:

- 1. Perforation of uterus
- 2. Uterine irritation → fibrous tissue
- 3. Retained placental fragments →
 - $-S_3$
 - Placental polyp
 - Malignant trophoblastic disease



Clinical picture

History

- Failure of placental delivery for ½ hour
- Bleeding →
 - . If the placenta is not separated at all....no bleeding
 - If it is completely separated.....minimal bleeding
 - If it is partially separated.....massive bleeding

Examination

- General → shock (hypovolemic ± neurogenic from Crede's method)
- Abdominal →
 - . Fundal level elevated above umbilicus
 - Signs of placental separation +ve or -ve
 - . Uterus may be atonic

Treatment

resuscitation -

Active 3rd stage management

- Ecbolics, massage, Brandt Andrews method →
- This will deliver an unadherent placenta in the absence of contraction ring

> If failed → Manual separation of placenta under anesthesia Introduce right hand along the cord, you may find

- Contraction ring
 - treat by delivering under GEA (halothane)
 - if failed → give uterine relaxant as amyl nitrite or other tocolytics
- Rupture uterus → laparotomy
- o Placenta adherent
 - reach the margin (line of cleavage between placenta & uterus)
 - take a fold of membrane, separate the placenta by sawing manner
 - placenta must be fully inspected for missing parts.

> If failed --> morbid adherence of placenta

- Supravaginal hysterectomy (ideal ttt)
- Conservation (if in much need of children & bleeding is not severe)
 - Cut the cord → leave the placenta or do morcellation
 - followed by → methotrexate, methergine, antibiotics
 - BUT still remains great hazard of hge & inf. →supravag.hyst.

K 4 DIC

(consumptive coagulopathy)



Definition

Paradoxical situation in which both thrombotic & fibrinolytic mechanisms are simultaneously activated → both <u>coagulation</u> & <u>hge</u> are present in the same time

Pathogenesis

- Presence of certain causes (severe / persistent) → activation of coagulation
- Dissemination of thrombosis → tissue ischemia & infarction
- Consumption of platelets / clotting factors + activation of fibrinolytic system → failure of clotting system → bleeding

Etiology

ФФ	Mechanism
 Placental abruption P.I.H. Amniotic Fluid embolism 	 liberation of tissue thromboplastin consumption in retromembranous hematoma endothelial damage → collagen exposure placental abruption may occur liberation of tissue thromboplastin procoagulant activity of amniotic fluid
4. Retained IUFD 5. Septic abortion (or any septic shock) 6. Induction of abortion	 liberation of tissue thromboplastin fibrinogen consumption in the dead fetus liberation of tissue thromboplastin release of bacterial endotoxin By intramniotic injection of hypertonic saline or urea → necrosis of uterus → thromboplastin
	 o forms complex with fibrinogen → inactive o esp.>5 unit or old blood → ↓coagulation factors o or massive hge & hypovolemic shock
8. Any excess tissue damage or hypoxia	- liberation of tissue thromboplastin e.g. placenta accreta, rupture uterus, hysterectomy

Clinical Picture

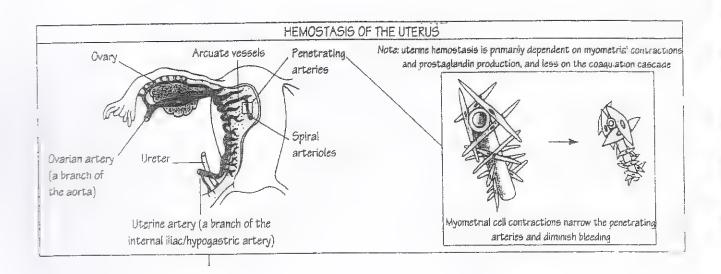
- Proper anticipation (presence of pdf e.g. abruptio placenta)
- Thromboembolism → pulmonary, renal, infarctions
- 3. Bleeding tendency → petechiae, <u>hematuria</u>, PPhge,

Coagulation factors

1 Plasma fibrinogen	VIII Anti-haemophilic factor
II Prothrombin	IX Christmas factor
III Tissue thromboplastin	X Stuart Prower factor
IV Calcium	XI Pl. thromboplastin antecedent
Pro-accelerin (labile factor)	XII Hageman factor
VII Pro-convertin	XIII Fibrin stabilizing factor

Pathways

Intrinsic pathway	Extrinsic pathway
Surface activation → collagen	tissue thromboplastin → activation
$XII \rightarrow XI \rightarrow IX \rightarrow X$	$VII \rightarrow X$
Assessed by PTT	assessed by PT



Investigations

→ Coagulation profile:

- 1. Platelet count (N: 250,000/ml). thrombocytopenia is <
- 2. Fibrinogen (N: 200-300mg% in pregnancy: 400-600mg%)
- 3. Fibrin degradation products (N: $10 \mu g/ml$) in DIC > $40 \mu g/ml$
- 4. D-dimers (in DIC >0.5 μg/ml)

→ Prolonged

- 1. Bleeding time (N: 2-4 min)
- 2. Clotting time (N: 6-12 min)
- 3. Prothrombin time (N: 12 sec.)
- 4. Partial thromboplastin time (N: 35-45 sec)
- 5. Thrombin time (time needed for conversion of fibrinogen to fibrin)

→ Weiner test (clot observation test...poor man's clot) Bed side test

- 5 ml blood in a test tube \rightarrow clot forms in 6-12 min & dissolves >30 m
- Failure of clotting within 15 min \rightarrow fibrinogen <100 mg%
- Early dissolution before 30 min → increased fibrinolysis

Treatment

[1] Treatment of the cause

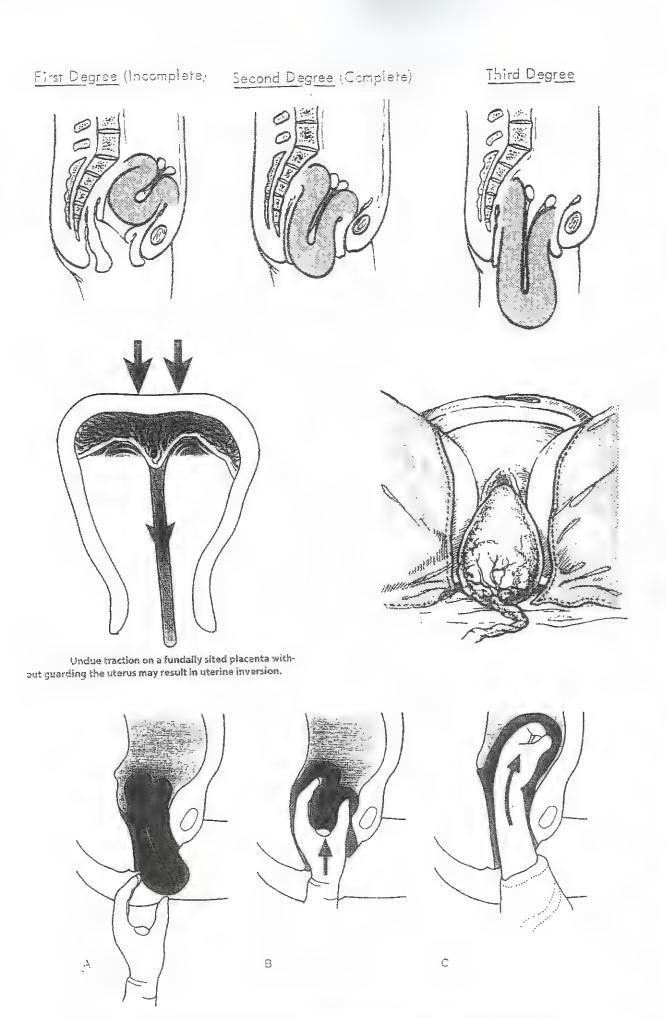
- o In most cases by termination of pregnancy as early as possible
- O Vaginal delivery is more <u>safe</u> although CS is more <u>rapid</u>

|2| Life saving measures

- Resuscitation
- Correction of coagulation defects
 - Fresh blood transfusion
 - Fresh Frozen Plasma (fibrinogen + coagulation factors)
 - Cryoprecipitate (dried fibrinogen or some coagulation factors)
 - Platelet transfusion

[3] Don't give

- Heparin → as it increases bleeding (except in IUFD: as there is intact vascular tree –the patient is not bleeding then heparin is stopped & TOP is induced after 6 hours
- Antifibriolytic drugs → as it increases thrombosis
 (they also cross placenta to the fetus)



Replacing an inverted aterus. (A) Reconstitution of interine inversion (B) Replacement of the interus forough the cervix (C) Restitution of the interus

Acute uterine inversion

Def: condition in which the uterus is turned inside out immediately after labor & before cx constriction (v.rare 1/3.000 - 1/30.000)

Degrees 1^{st°} → just cupping of the fundus

 $2^{nd^{\circ}} \rightarrow$ inverted fundus protrudes through the cervix into vagina

 3^{rd} \rightarrow inverted fundus appears outside the vulva (the <u>complete</u> type)

Etiology

Iatrogenic (bad 3rd stage management) //

- 1. Crede's method while uterus is lax
- 2. Cord traction while placenta not yet separated
- 3. Manual removal of placenta esp. if it is adherent

Spontaneous = precipitate labor or excessive straining



Clinical Picture

→ History

- * severe lower abdominal pain with continuous bearing down
- * Fullness (2nd) in or something protruding (3rd) from the vagina
- * PPhge (atony) may be minimal if
 - Placenta is still attached
 - . In severe degrees with kinking of blood vessels

\hookrightarrow Examination

- * General → Shock (hypovolemic & neurogenic)
- * Abdominal: . 1st degree → cup shaped fundus $\cdot 2^{\text{nd}} \& 3^{\text{rd}} \rightarrow \text{absent fundus}$
- * Vaginal
 - 1st → depressed fundus inside the uterus
 2nd → cervix surrounds inverted fundus

 - $3^{rd} \rightarrow$ fundus protrudes through the vulva

Differential Diagnosis

- 1. Causes of postpartum shock
- 2. Uterine prolapse (the cervix –external ostium– is found)
- 3. Fibroid polyp (uterine sound passes all around)

Treatment

- Prophylaxis = avoid pdf. + proper 3rd stage management
- > Active => Resuscitation + Manual reduction (or hydrostatic ") Under GEA (halothane / amyl nitrite / tocolytics) **
 - First reposit the uterus then → remove the placenta ^x
 - Then \rightarrow ecbolics + massage
 - Then → Pack + antibiotics

Amniotic fluid embolism

Incidence > 1/30.0000 with 50% mortality

Etiology

- o AF may enter into the maternal circulation d.t.:-
 - 1. Increased intrauterine pressure

accidental hge, oxytocin overdose with intact membranes

2. Opened uterine or endocervical veins

as in genital tract lacerations e.g. rupture uterus

The above factors also lead to fetal distress → meconium stained AF →
this potentiates the toxic nature of AF → worsens the symptoms

Pathogenesis

- o Immediately or shortly after a difficult delivery:-
 - RDS & circulatory collapse (extensive pulmonary vascular obstruction d.t. the AF particulate matter → acute cor-pulmonale → abrupt hypoxia & CHF)
 - DIC → bleeding from genital tract & all other sites of trauma
 - Deep coma & immediate death (>50%)
- c Recently, it is proved to be a form of <u>anaphylactic</u> shock to the antigenic AF (thus AF embolism is a mis.....)

Diagnosis

- \circ Suspected in \rightarrow any case of sudden postpartum collapse & DIC
- Proved by → finding AF debris (fetal squamous cells, lanugo hair, vernix) in the pulmonary vessels by <u>autopsy</u>[±]
- Investigations → ECG, chest X-ray, V-Q scan

Monogement

- Very difficult (serious > pulmonary embolism) → only few cases succeed
- Immediate transfer to ICU > − Cardio-pulmonary support
 - Management of DIC
 - Corticosteroids
 - Monitoring different organs

Sudden postpartum collapse



Non-obstetric causes

- 1^{ry} PPhge:- rupture $\sqrt{\sqrt{,-,-,-,-}}$
- Tringe.- Tupture V V,-,-,-,
 Eclampsia
- Pulmonary thrombo-embolism
- Amniotic fluid embolism
- Cardiogenic e.g. peripartum cardiomyopathy
- Cerebrovascular accidents
- Anesthetic complications e.g. Mendelson \$
- Anaphylactic shock



Resuscitation. A. Algorithm. B. Position for cardiopulmonary resuscitation.

Shock in obstetrics

Definition = a state of circulatory failure

(hypotension, tissue hypo-perfusion

Etiology

- O Halc shock bleeding in early preg., APHge, PPHge
- O Neurogenic pain in early preg., pain in late preg.
- O Pul. embolism amniotic fluid or thrombus
- O Splanchold sudden drop of intrauterine pressure (polyhdramnios, twins)

Clinical picture

- History suggestive of
 - Etiology e.g..missed period + acute abdomen → disturbed ectopic
 - Pdf e.g.....preg comp (anemia, PIH)..labor comp (prolonged / obstructed)
-) Examination
 - 1. General → shock:- low B.Pr., subnormal temp, rapid weak pulse, pale cold clammy skin, peripheral cyanosis, oliguria
 - 2. Abdominal
 - T, R, RT → internal hge e.g. ectopic
 - Bilateral adnexal swellings → V.mole
 - 3. Local
 - Offensive discharge → sepsis
 - Vaginal bleeding → hgic

Treatment

- > General
 - Intravenous cannula..... Analgesia (morphia 15mg IV)
 - Raise legs.....O₂ inhalation.....Warmth (but not direct, to avoid VD)
- Monitoring (by fluid input & output chart)
 - Catheterization → urine should not be < 30 ml/hr
 - CVP \rightarrow kept between 8–12 cm H₂O
 - Replacement → start by available fluids till blood is ready
- > Drugs
 - Vaso-pressors ± inotropics
 - Corticosteroids, correction of acidosis (Na bicarb)
 - Antibiotics (in septic shock)
- Special
 - Disturbed ectopic....laparotomy & salpingectomy
 - Acc.hge.....TOP better vaginally
 - Rupture uteruslaparotomy & supravaginal hysterectomy

Obstetric trauma

> Types

0 Maternal

- * Genital tract trauma
 - Tissue lacerations (perineal, vaginal, cervical, uterine)
 - e Hematoma formations (vulval, vaginal, broad ligamentary)
 - c Tissue necrosis (bucket handle tear of cx, necrotic fistulas)
- * Mon-genital tract trauma (usually d.t. forceps)
 - Injuries of pelvic joints & bones → rupture SP, coccyx, sacro-iliac lig.
 - Hematoma of → rectus abdominis muscle
 - Due to rupture of the superior deep epigastric vessels
 - More common in MP after strenuous labor efforts
 - May occur after cesarean section
 - $C/P \rightarrow$ sudden severe pain \pm shock

@ Fetal (esp in breech)

- * Herd injury (ICHge, fractures of the skull)
- * Proportion with nerve (brachial plexus, facial, phrenic nerve palsy)
- * Minor is restal (fracture clavicle, other long bones)
- * Yoft tissue (sternomastoid, head, abd organs lacerations)

> Long term sequelas

Vagina	Cervix	Uterus	Levator ani
Dyspareunia	chronic infection	hysterectomy	Prolapse
Fistula	Infertility	rupture uterus in	Incontinence
	PIO, PTL	next pregnancy	- urinary
1	cervical dystocia	ureteric comp.	- anal

Comp. of 3rd stage of labor



> Short term

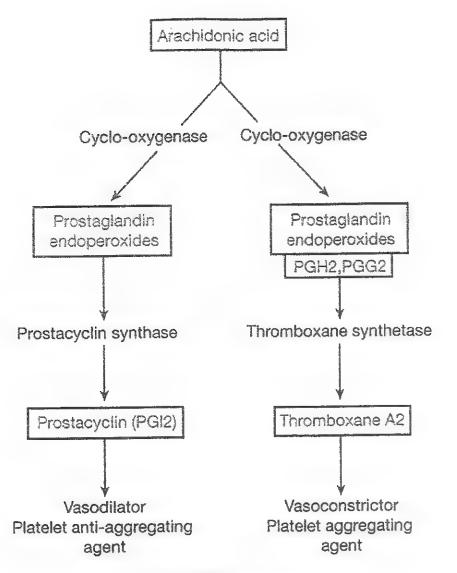
- Causes of PPhge. including 3rd stage hge (retained placenta)
- Shock & its complications → acute renal failure & DIC
- Amniotic fluid embolism
- Complications of anesthesia & blood transfusion

> Lingiermi

- On puerperium $\rightarrow 3$ S
- Infertility → due to hysterectomy or Ashermann syndrome
- Prolapse & incontinence (urinary & rectal)

Pre-eclampsia
Diabetes milletus
Heart Diseases
Hyperemesis gravidarum
Urinary tract infection
Anemia
Thromboembolism
Thyroid disease
Respiratory disease
Surgery & Pain

Choloida Abnomici Pregnancy Dreckes Wifin Pregnency



Prostaglandin metabolism.

} may occur } in the 1st ½

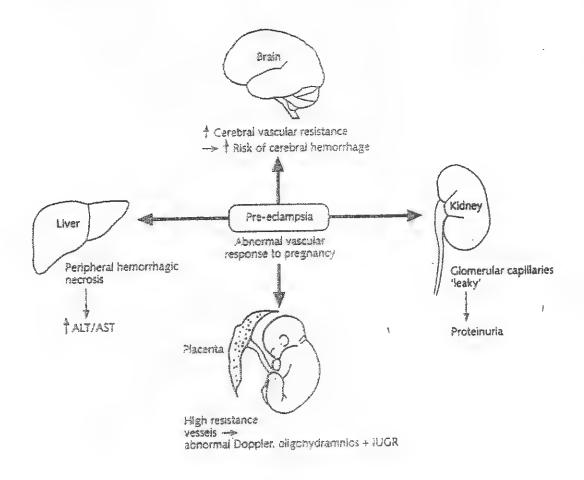
of preg

🖆 HTN disorders: [1] Pre-eclampsia 👙
Occurrence of Hypertension, Proteinuria, pathological Edema . In the 2 nd half of pregnancy in a previously healthy woman . Mainly affecting PG
 PIH ⇒ Pregnancy Induced Hypertension (toxemia is a misnomer; PET x) EPH-gestosis ⇒ Edema, Proteinuria, Hypertension (by gestosis organization)
It is the commonest / medical disorder in pregnancy (5 - 10%)
Etiology
[1] Prostaglandin imbalanee: \downarrow prostacyclin, PG-E ₂ &nitric oxides (EDRF) ↑ thromboxane * & PG-F ₂ &fibronectin
[2] Renin-angiotensin activation \rightarrow with \uparrow sensitivity to angiotensin II \searrow severe VC & aldosterone secretion
[3] New theories
- Immunological → abnormal immune response to invading trophoblast & fetal antigens (∴ it is > in PG)
 Genetic predisposition → multifactorial? recessive?
[4] Older theories XX
- Dietary factors
Predisposing factors
○ Patient ccc ⇒ . Extremes of age (<20 or >35)
(low socio <u>PG</u> exclusively (however it may occur in MG) economic . Obesity
class) .+ve family history
Obstatric disorders = PIH is more common when there is large volume of chorionic tissue. Twins } It even

. Hydrops fetalis ++ APS \sim Medical disorders \Rightarrow D.M. \sim , chronic hypertension, chronic nephritis, SLE

. Polyhydramnios

. Vesicular mole



End-organ effects of pre-eclampsia.

Diagnosis of HELLP Syndrome

Hemolytic anemia
Schistocytes on peripheral blood smear
Elevated lactate dehydrogenase
Elevated total bilirubin

Elevated liver enzymes Increase in aspartate aminotransferase Increase in alanine aminotransferase

Low platelets
Thrombocytopenia

Main pathology

- Vasospasm^x + endothelial cell injury → hypertension + hypoxic injury → degeneration of cells & hge
- Multiple organs are involved ∴it is a **syndrome** (not a disease) and.....HYPERTENSION.... is the *milestone* of this syndrome

Complications

.....the 2nd cause of MMR.....

1 Maternal

> Immediate

- CNS → GODampson, cerebral hge / infarction, cerebral edema
- Retina \rightarrow papilledema & retinal hge (stellate) \rightarrow detachment
- CVS → hypertrophy of heart (cardiomegally)
 . Up to acute HF
- Resp → laryngeal edema, pulmonary edema
- Liver → . Mainly periportal necrosis → Jaundice
 Subcapsular (Glisson capsule) hge & rupture
- Kidney \rightarrow

Proliferation of endothelial, epithelial, mesangial cells \rightarrow narrowing of the glomerular vessels $\rightarrow \downarrow$ RBF & \downarrow GFR

- * Damage of glomeruli → proteinuria → edema
- * Two major complications may occur
 - . Renal tubular necrosis (reversible)
 - . Renal cortical necrosis (irreversible)
- Adrenal → acute adrenal failure → Addisonian crisis
- Metabolic →
 - * Salt & H₂O retention
 - * Haemoconcentration " (\frac{1}{2} intravascular volume ")
 - * WELLP syndrome in severe cases 💸

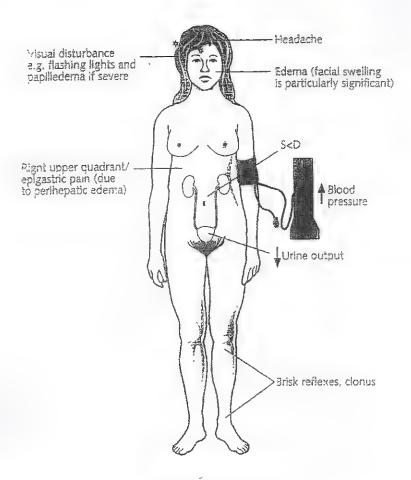
→ Hemolytic anemia, Elevated Liver enz., Low Platelet

> Remote

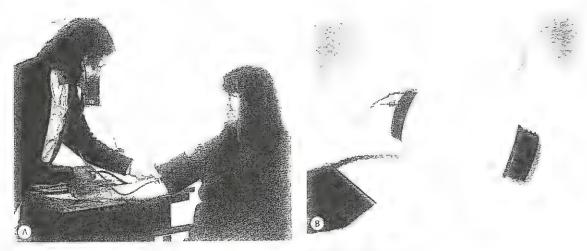
- Residual hypertension or proteinuria (5–10%)
- Recurrence (MG) 30-50%

Petal & placental

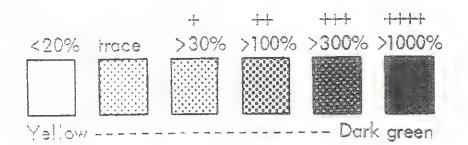
- ➤ Normally → the trophoblast invades the media of the spiral vessels of decidua at 20 weeks (2^{ry} wave of trophoblastic invasion)
- ▶ In PIH \rightarrow this is absent \rightarrow the media persists \rightarrow ↑ vascular resistance
 - y * DUGR & IUFD
 - * PTL (idiopathic or iatrogenic/)
 - * Abruptio placenta ightarrow DIC



Signs and symptoms of pre-eclampsia.



Early detection of pre-eclampsia is Important. (A)
Measurement of blood pressure (reproduced with permission)
(B) Testing for urinary protein.



Clinical picture

Symptoms:...

ONLY IN SEVERE CASES

1] NEUROLOGICAL SYMPTOMS:

- Headache (frontal, persistent, not responding to analgesics)
- Nausea & vomiting
- Visual disturbance as blurring of vision up to ↓ visual acuity
- 2] EPIGASTRIC PAIN → stretch of liver capsule (or subcapsular hge)
- 3] OLIGURIA (<400 ml /day) & ANURIA (<100 ml /day)
- 4] SYMPTOMS OF ANY COMPLICATION e.g. HF & Pulmonary edema

➤ Signs: MORE IMP. & PRESENT < SYMPTOMS</p>

1] Hypertension

- Systolic ≥ 140 mmHg or ↑ 30 mmHg over previous value
- <u>Diastolic</u> (✓) ≥ 90 mmHg or ↑ 15 mmHg over previous value Measured at semisittingor left lateral position

2] Proteinuria /

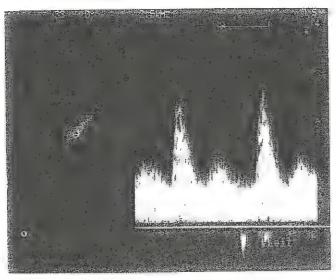
- Non-selective
 - . A serious sign → glomerular damage
 - . Detected by \rightarrow albustix
- Significant if \geq 300 mg/dl (1+). normally ≈ 150 mg/dl -
 - More accurate if measured in 24 hours collected urine
 - > Recently spot protein / creatinine ratio in a urine sample
 - 🖫 Proteinuria without HTN is also risky for both M 🗟 F

W Mono-symptomatic gestosis (presence only of hypertension or proteinuria may occuri

3] Edema

- Occult = detected by rapid gain weight > 1 kg (2 pds) / 2 wks (Normally $\rightarrow < \frac{1}{2}$ kg/wk in 2nd & 3rd trimesters)
- Manifest dorsum of foot, shin of tibia (m.b. normal) then become non-dependant → vulva → worsens → abdominal wall (Peau d'orange') → swollen fingers (rings become tight) → puffy eye lids & papilledema. The worst is P.edema
- <u>∃bsent</u> = dry pre-eclampsia (the worst \$)

YEdema is d.t. (cap. damage, hypoproteinemia, hypertension) \searrow Edema is not essential for diagnosis $\bar{\otimes}$ has little prognostic value



Uterine artery Doppler notching at 24 weeks is predictive of pre-eclampsia and intrauterine growth restriction in high-risk mothers.

Differential diagnosis from other causes of

Edema

	SEASON OF THE PROPERTY OF THE
Bilateral	Unilateral
- Physiological (at feet & ankle only)	- DVT
- Generalized anasarca e.g. H.C.R.N.A	- Varicose veins
- Endocrinal e.g. Cushing, myxedema	- Lymphedema

> Hypertension

- * Pregnancy INDUCED hypertension
 - . PET (PIH)
 - . Eclampsia = (PET + convulsions)
 - . Gestational (transient) hypertension is the *appearance* of hypertension for 1st time > 20 weeks in *absence* of proteinuria & edema. It usually disappears > delivery.
- * Pregnancy ASSOCIATED (coincidental / chronic) hypertension
 - . HTN ($\geq 140/90$) present ≤ 20 weeks, or
 - . HTN 1st diagnosed in pregnancy & persists after puerperium
- * Pregnancy ACCRAVATED hypertension
 - . Super-imposed PET (occurrence of PET on top of chronic HTN)
 - . Super-imposed eclampsia (occurrence of ecl. on top of chronic HTN)

> Proteinuria

- * False proteinuria (the commonest cause 🗸)
 - \(\text{contamination from vaginal discharge; avoided by:} \)
 - Mid-stream urine sample (MSU) i.e. clean catch technique
 - Catheter specimen x (not preferable)
- * Urinary tract infection
- * Orthostatic (appear at end of day) -> pr from lumbar spine on Lt renal vein

Investigations

- 1. Renal function tests: uric acid $(1^{st} to^{\uparrow x}) \rightarrow$ creatinine, urea
- 2. Liver function tests
- 3. $CBC \rightarrow Hct, HELLP$
- 4. *Coagulation profile* → DIC (platelet count, antithrombin III)
- 5. Fundus → spasm, haemorrhage, exudate, edema
- 6. ► Fetal → FWB ✓
- * Tests to detect possible development of PE [SCREENING]:
 - . Doppler \rightarrow high vascular resistance $\checkmark \checkmark$...early diastolic notch
 - . Roll over test († BPr in supine position > 20 mmHg) 🦻
 - . Cold water immersion test († diastolic pr >20mmHg) 🦻
 - . Angiotensin II infusion test 🖓
 - . ↑ plasma fibronectin, ↓ urinary calcium 🔊

Classification

- ♦ Pre-eclampsia may be mild or severe if:-

 Ø

 Ø
 - Signs * B.Pressure [Systolic ≥ 160 mmHg Diastolic ≥ 110 mmHg] * Proteinuria ≥ 500 mg/dl (++) or ≥ 5 g/L/24 hr collected urine *
 - Symptoms -> appearance of any symptom esp;.... oliguria or anuria
 - Complication → Maternal (HELLP, DIC) or Fetal (IUGR)
 - Investigations → denoting any organ damage
- Fulminating Preeclampsia: (impending eclampsia)
 - Severe PE (symptoms, severe proteinuria) + Hyperreflexia
 - If left → may develop eclampsia [therefore ttt as eclampsia]

Treatment (1)

₱ Prophylaxis ₱

- \circ Early detection by regular ANC \checkmark \rightarrow BPr., albumin, screening tets (esp for HRG)
- Anti-platelets → as low dose aspirin (75mg) or juspirin (81mg)
- May give \rightarrow vit E (anti-oxidant π), omega 3 (fish oil).

9 Mild cases 3

- 1] If mature Terminate
- 2] Otherwise Conserve
 - * Bed / Mental rest -> sedatives in extreme cases...e.g diazepam (5 mg day)
 - * Diet → balanced i.e. → avoid excess [salt, fats, CHO], not salt restriction
 - * Antihypertensives (some say no need: mild case 🔊)

	Action	Dose
α-Methyldopa (Aldomet)	Central action a	250 mg 1x4
The most safe & widely	(acts as a false transmitter in the	-up to-
used in mild cases 🗸	brain $\rightarrow \downarrow$ noradrenaline).	2 –4 gm /day
β -blockers (alone or + γ)	wood with continue to the language	10 mg
Nefidipine (Adalat)	used with caution \rightarrow it \downarrow placental flow & FWB (used with caution)	up to 40 mg /day

* Observation

- Daily → FHS / 6 hrs.....BPr......Albuminuria
- Weekly → FWB......RFT, LFT, fundus...weight (for edema)
 - > Corticosteroids may be given to enhance lung maturity 🔊
- > Conservation is continued \$15 till maturity (37-8 wks) unless }
 - Disease → <u>severe</u> PET
 - Mother → distress e.g. HELLP syndrome
 - Fetus → distress e.g. apnormal CTG "UGF

Severe cases...TOP

....The only cure
in spite of fetal maturity...

[1] Hospitalization

⇒ Eclampsia room or Obstetric-ICU

Patient lies on her side in a semi-dark quiet room with available \rightarrow O₂ supply, mouth gag, tongue depressor, endotracheal tube, suction machine, anticonvulsant drugs started immediately

> Observation for

- Vital signs → BP, P, T, respiratory rate
- Level of consciousness
- Fluid intake & urine output → chart
- During fit (emergency ttt...even done at home) → insert mouth gag, avoid biting tongue, place on her side to prevent aspiration

[2] Anticonvulsant therapy (to control & prevent further fits)

Magnesium sulfate [MgSO4.7H2O]

* Route

- IV: 1/4-6 gm slowly (over 15-20 m) then.....1-2 gm/hr by drip
- IM: loading 14 gm (4 IV + 10 IM -5 gm/ buttock-).... then 5 gm / 4 hrs

 > better avoided → sterile abscess / very painful

* Action

- Peripheral skeletal muscle relaxant (↓ A.Ch & Ca⁺⁺ at NMJ) ✓
- MILD Subcortical depressant
- MILD Transient hypotensive effect [vasodilator + diuretic]

* Toxicity Signs

- Absent knee reflexes.....8–12 mEq/L

- On high level ______ neonatal resp. depression



Due to this narrow safety margin (4-7 mEq/L), the following must be checked before each dose 3

- ~ Knee jerk (patellar reflex) is still present
- ~ Respiratory rate not < 16 /min
- ~ Urine > 30 ml / hr (the only way for excretion)
- ~ Or the best → measure serum Mg level
- * Duration = continue therapy for 24-48 hrs after <u>delivery</u> or the last <u>fit</u>
- * Antidote Ca⁺⁺ gluconate slowly 10ml 10% solution ± O₂ ± intubation
- * In resistant cases Phenytoin...or...Pentothal Na (Intraval)

[3] Antihypertensive therapy

o *The aim is* to prevent maternal intracranial hge or HF; but keep diastolic BPr between 90-100 mmHg (to avoid ↓ placental bl. Flow → IUFD) 1

	Action	Dose	
Hydralazine (Apresoline) Drug of choice in Severe cases **	Direct arteriolar VD ↑ COP, ↑ renal flow	10 mg IV bolus	
Labetalol (Trandate)	α and β blocker It crosses placenta → fetal bradycardia. Also contraindiintervals cated in pts with 1 ^{sto} HT block		
Nefidipine (Adələt) Sublingual	Potent drugs with rapid action → sudden severe hypotension → life threatening for mother (cerebral hypo-perfusion) & fetus (↓ utero-placental flow). Diazoxide also causes neonatal hyperglycemia		
Diazoxide (IV) most dangerous			
Others	 ○ Nipride (Na <u>ni</u>tro<u>pr</u>uss<u>ide</u>) ◇ Tridil (Ni<u>tr</u>oglycer<u>i</u>ne) 		

○ Piasma volume expansion ⇒ given with extreme caution (volume overload)
 ○ NO Diareties ⇒ ↑ hemocone. & electrolyte imbalance (except in HF or P.edema)

[4] Termination ///

- Induction / augmentation of labor: ►+ if delivery is expected soon
 By AROM & syntocinon
 - ! Muge → intrapartum continuous fetal monitoring
 - 2^{-1} stage \rightarrow shorten 2^{nd} stage by forceps
 - Single avoid ergometrine after delivery (oxytocin may be given)
- * Casarean section; > but first correct the general condition (anti-HTN, MgSC4, correction of the severe metabolic acidosis due to fits)

[5] Treatment of complications

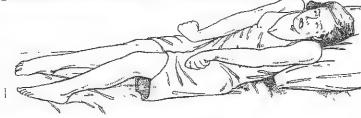
- Maternal.....renal shut down, HELLP
- → Fetal.....IUGR

[6] Postportur

- Vaticer vulsant therapy continued for 24-48 hrs after....
- Antihy ercensive therapy may be given if needed
- Newson; for PIH in next pregnancy (became high risk)

Eclampsia A

Definition = occurrence of fits (grand-mal-seizures ") in a patient with PET



Etiology

- Cerebral irritation by edema or electrolyte imbalance (\(\)Na\(^+ \)
- Cerebral ischemic foci by vasospasm or platelet thrombi

Stages of fits

1] Premonitory / Prodroma (3-5 min)

- . Twitches in muscles of eyes or face, rolling of eyes
- . Severe headache, disturbed consciousness
- 2] Tonic phase (30 sec)

All muscles of body pass into spasm. Back is arched (episthotonos), limbs stretched, respiration stops → cyanosis

31 Clonic phase

Intermittent contraction & relaxation of muscles \rightarrow biting of tongue, vomiting, aspiration, spontaneous defecation or micturition, stertorous breathing, falling from bed \rightarrow fractures

4] Coma stage (d.t. severe acidosis)

Variable \rightarrow may recover <u>OR</u> pass into another fit (recurrent or status eclampticus) <u>OR</u> dies without recovery

> Types of fits

Antepartum colampsia ightarrow 70% "

Intrapartum selampsia ightarrow 20%

Postpartum aclampsia $\rightarrow 10\%$ (worst $\stackrel{?}{\Rightarrow}$), during 1st 48 hours up to.... (the disease process is continuing though pregnancy has ended)

Differential diagnosis

Convulsions

- Cerebral: epilepsy (similar!!), ICHhge, infection, tumor, trauma
- Metabolic. hypo- or hyperglycemia, hypocalcemia (tetany) / Tetanus
- Poisoning by strychnine
- Hysterical ✓

Coma

- Cerebral.....poisoning
- Organ failure as uremia or hepatic failure

Complications $\Phi \Phi \Phi$

O Maternal (MMR => 10%)

> Complication of convulsions

- ☆ Asphyxia due to
 - Tonic contraction of respiratory muscles
 - Inhalation of vomitus
 - Inhalation of blood from bitten tongue → aspiration pneumonia
 - Tongue falls backwards
- ☆ Severe metabolic acidosis
- ☆ Hyperpyrexia

> Complications of PET

- ☆ Organ failure e.g. heart, renal, suprarenal, hepatic failure
- A Haemorrhage in vital organs e.g. IChge, abruptio placenta

② Fetal (high PNMR ⇒ 30%)

- ➤ TUGR (placental insufficiency)
- ▶PTL (spontaneous or iatrogenic)
- ▶ IUFD (accidental hemorrhage, maternal hypoxia in fits)

Bad prognostic signs (Eden's criteria)

- 1] Fits \rightarrow recurrent (esp >6) & postpartum
- 2] Coma \rightarrow long (esp > 6 hrs) & deep
- 3] Vital data * BPr: Systolic ≥ 160 mmHg Diastolic ≥ 110 mmHg * Pulse > 120 Temp > 38°c......RR > 40 /min
- 4] Oliguria, Anuria
- 5] Dry eclampsia
- 6] Organ damage \rightarrow HELLP syndrome (olive green jaundice). S&S

Managementthe same lines as in severe PET.....

- Eciampsia room....
- Drugs
 - Anti-hypertensives
 - Mg-SO4
- Examination ⇒ TOP
 - If favorable (Bishop > 8) \rightarrow induce labor
 - If not favorable (Bishop ≤ 8) \rightarrow CS (after correction of acidosis)
- · Care of complications
 - Maternal
 - Fetal

Classification	Nursing responsibilities	Obstetricians' treatment
Potential PIH	Report significant rise in blood pressure, or excessive weight gain, to obstetrician.	Usually no treatment required. See patient in 7 days.
Mild PIH	Report rise in blood pressure or excessive weight gain to obstetrician.	Possible admission to hospital, depending on socio-economic conditions. If not admitted, see patient in 3 days.
Moderate PIH	In hospital: Four-hourly recording of the blood pressure. Twice-daily urne testing for protein. Regular observation of the patient's condition, including fluid intake and output. Bed rest, but toilet privileges allowed.	Admit to hospital. Sedation (if indicated). Labetalot (starting) 100mg twice daily or atenoic (starting) 100mg in evening or exprendiol (starting) 20mg three times daily on methyldopa (starting) 250mg three times daily. Nifedipine 10mg sublingually repeated as needed.
Severe PIH	Two-hourly blood pressure recording for 6 hours, then 4-hourly. Urine testing for protein and acetone twice daily. Fluid intake and output recorded. Careful observation of the patient for the signs of imminent eclampsia. Complete bed rest for 24 hours, thereafter possible toilet privileges.	Admit to hospital. Depending on the severity of the illness give: a) Magnesium sulphate (see page 126). This is the preferred medication b) Hydralazine intravenously Intravenous frusemide 20 mg or stable plasma protein substitute (SPPS), if persistent oliguria. !Caesarean section.
Imminent eclampsia	The patient requires careful systematic observation as eclampsia is a possible outcome. The blood pressure requires frequent estimation, at intervals determined by the obstetrician. Fluid intake and urinary output must be measured meticulously, and the unne tested quantitatively for protein.	Magnesium sulphate (see page 126). This is the preferred medication. Hydralazine intravenously. Caesarean section.

[2] Chronic hypertension

Definition = presence of HTN < pregnancy...or...< 20 wks

- Primary (essential) ✓✓
- Secondary e.g. Renal, Pheochromocytoma, Cushing syndrome, Conn's disease, Coarctation of aorta, Thyrotoxicosis

Clinical picture

- * PDF
 - Old age, high parity, +ve family history
 - Cardiovascular risk factors: smoking, †plasma lipids, obesity, DM
- * SYMPTOM-LESS. Ask for)
 - Duration of hypertension before pregnancy & ttt received
 - Previous complications
 - . Medical: stroke, myocardial infarction
 - . Pregnancy: superimposed PET, IUGR, IUFD
- * SIGNS
 - Hypertension:
 - 1) < 20 weeks, or
 - 2) if it persists after puerperium

Grade	Diastolic BPr
Mild	90 – 105
Moderate	105 – 115
Severe	≥ 115

- No edema or proteinuria (unless complicated by superimposed PE or HF)

Investigations

-reflect chronicity......
- ECG changes & cardiomegally
- Renal functions → creatinine clearance
- Fundus → atherosclerosis + hge

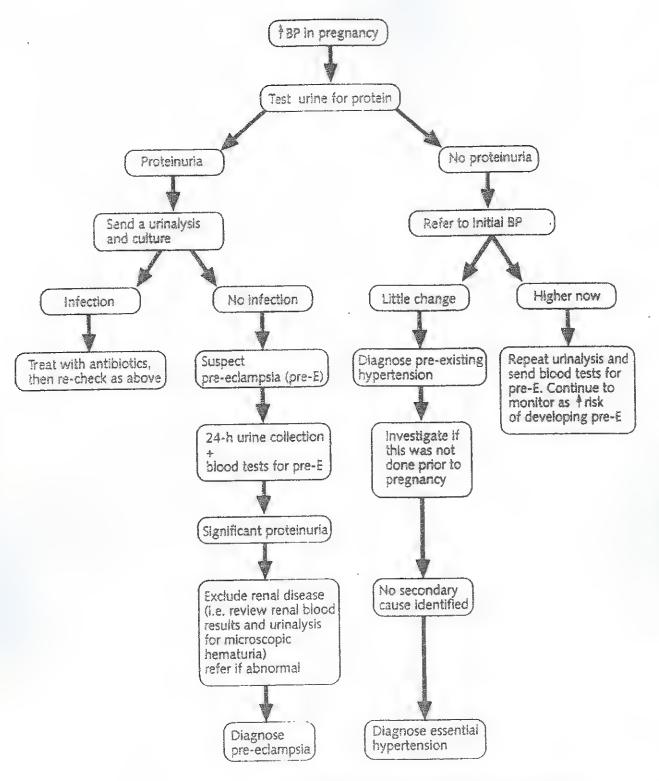
Complications

[1] Effect of pregnancy = hypertension

- * Increased severity
- * Superimposed PE when >
 - Blood Pressure (S: > 30 mmHg / D: > 15 mmHg)
 - Proteinuria ± Edema
 - Appearance of complications *peculiar* ✓ ✓ to PET (M. or F.)
 - Lower 24 hr urinary Ca⁺⁺ (40 mg) than in chronic HTN (220 mg)

[2] Effect of hypertension => pregnancy

- * Maternal → all complications of PE esp Accidental hge.
- * Fetal → IUGR, IUFD, PTL



Algorithm for hypertension in pregnancy.

Treatment

> Mild => conservation

☆ Antihupertensives

- ² 1st choice → Methyldopa: check liver enzymes /trim. (liver affection)
- 2nd choice → Hydralazine
 - .Tachyphylaxis *
 - . If $> 200 \text{ mg/d for} > 6\text{m} \rightarrow \text{lupus like syndrome}^{\text{m}}$, fluid retention
- 3 3rd choice → Labetalol (200 mg tab 1x3 up to max 1.6-2.4 gm/d)

☆ Used with coution

- Nifedipins... safe, but may → acute hypotension
- **Clonidine**....safe, but acute withdrawal → hypertension

☆ Contraindicated

- ACE inhibitors....fetal renal failure **
- Diuraties... except in severe cases (heart failure)

> Severe

- Medical treatment → good response → continue
- Failed medical control → terminate

	PET	Chronic HTN	Chronic nephritis	
Incidence	75%	20%	5%	
Past history	– ve	< pregnancy	< pregnancy	
Hypertension	> 20 wks	< 20 wks	< 20 wks	
Edema	+ ve	– ve	+ ve	
Proteinuria	+ ve	Ve	+ ve	
ECG changes	- ve	+ ve	- ve	
Renal function	↓ if severe	affected with time	impaired	
Fundus	↓ if severe	sclerotic with time	albuminuric nephritis	
TTT	TOP if severe	according to degree of M & F affection		
Sequise	Recur in 30%	condition persists & usually deteriorates		

Designation Chronic metabolic disorder of CHO metabolism

Due to <u>absolute</u> or <u>relative</u> decrease in insulin

in response to CHO challenge → hyperglycemia

* BIPHASIC GLUCOSE CONTROL IN PREGNANCY

- C FIRST HALF OF PRESNANGY
 - ⇒ improved glucose tolerance * due to:
 - 1 ed insulin response to a glucose load
 - ↓ gluconeogenesis & ↑ glycogen deposition
- D SECOND HALF OF PREGNANCY
 - ⇒ increased insulin resistance *, therefore:
 - 90% of DM with preg are GDM
 - GDM is not clinically apparent untilwks
 - : Screening is better done at this time
 - ∴ Patients with GDM escape CFMF

INCOMPANION (PED)

1) <u>According</u> to onset

	Type I (10%) (Juvenile onset, IDDM)	Type II (90%) (maturity, non IDDM)
Etiology	Autoimmune	Familial tendency
	(island cells anti-	(complex & multi-
	-bodies e.g. viral inf.)	-factorial etiology)
Insulin	↓ (defect in pancreas)	↑ (insulin resistance)
Weight	Thin	Obese
Comp	More e.g. DKA	Less

2) According to stages °

- » Potential Diabetes patient is not diabetic but there is 1ed risk esp. if:-
 - Positive family history (parents or her twin)
 - Previous delivery of macrosomic or malformed fetus
 - Parity (GMP ≥ 5) or obesity (>120% of ideal body weight)

> Lacent Diabetes

The patient is not diabetic but on exposure to stress or corticosteroids --> GTT is diabetic (:: GDM is one of its forms)

- > Coemical Digoetes asymptomatic patient but GTT is diabetic (IGT)
- > <u>€x 11 space d'onneces</u> → clinical diabetes (all < that is the Pre-Diabetic state)

Complications of D.M



Effects of pregnancy D.M. D.M. Effects of pregnancy D.M. D.M. Effects of pregnancy Effects of pregnancy D.M. D.M.

- O Potentially diabetogenic + worsens established D.M. due to
 - Anti-insulin hormones (HPL, E, Pr., corticosteroids, prolactin)
 - Insulinase activity in placenta 🖓
- Praexisting complications may become aggravated with increased liability for DKA
- Difficult control with liability to hypoglycomia during:
 - D PREGNANCY
 - Renal glucosuria (↑RBF→↑GFR→↓renal threshold to 150 mg%)
 - Alimentary glucosuria
 - Morning sickness & vomiting → starvation ketosis
 - Glucose passes to the fetus by facilitated diffusion... *
 - □ LABOR (d.t. uterine activity)
 - D PUERPERIUM
 - Loss of placental hormones
 - Breast uses glucose to produce *lactose*

₩ Effect of D.M. = pregnancy

O Maternal

- 3 PREGNANCY
 - Preeclampsia.....in 25% * (vasculopathy)
 - Polyhydramnios in 25% of cases

(large placenta....fetal polyuria....Anencephaly)

- Preterm labor....overdistension d.t. macrosomia & polyhdramnios
- Placenta previa & abruptio placenta (PIH)
- Pyelonephritis (recurrent) liability to infections as candidiasis

3 PARTURITION

- PROM → fetal & maternal infection
- Prolonged labor (d.t. macrosomia → obstructed labor → rupture ut.)
- 3 PUERPERIUMS
 - Postpartum hemorrhage (atonic, traumatic)
 - Puerperal sepsis
 - Pulmonary embolism (obesity, vasculopathy, difficult labor)

Congenital anomalies in infants of diabetic mothers

Cardiac

- · atrial septal defect
- •ventricular septal defect
- · coarctation of aorta
- transposition of great vessels

Other

 single umbilical artery

Gastrointestinal.

- · anorectal atresia
- · duodenal atresia
- tracheo-esophageal fistula

Skeletal and central nervous system

- anencephaly
- caudal regression syndrome (very rare, but highly specific for diabetes mellitus)
- · microcephaly
- · neural tube defects

Renal

- · hydronephrosis
- · renal agenesis
- ·ureteral duplication
- · polycystic kidneys

O Fetal

- ➤ Abortion..... ↑ 3x (if uncontrolled DM) how?
- - Especially if HbA_{1c} is increased
 - The commonest are:
 - . CVS \checkmark (10x) \rightarrow VSD, transposition of great vessels, coarctation of aorta
 - CNS $(5x) \rightarrow$ anencephaly, spina bifida, meningocele
 - . GIT ... renal ... skeletal
 - A <u>rare</u> but very specific (pathognomonic) malformation is *caudal* regression syndrome (sacral agenesis **). This is disproved now.
- > IUGR in 20% of cases (placental insufficiency d.t. the vascular changes)
- > Fetal macrosomia (40% of cases)
 - Due to increased glucose in mother → hyperglycemia in the fetus
 → ↑ insulin from fetus → islet cell hyperplasia → marked anabolic effect. There is associated stimulation of adrenal cortex → ↑ed steroids → Na & H₂O retention
 - Newborn is large heavy plethoric fatty with cushingoid features
 - All this makes the baby more liable to all listed complications.....
- > IUFD due to
 - Hyperglycemia ± ketosis or Hypoglycemia
 - Vascular affection → chronic placental insufficiency & PIH
 - Congenital malformation
 - Unexplained sudden IUFD (usually after 36 weeks, repeats at same time)

® Neonatal ФФ

- 3 → RDS (insulin antagonizes action of corticosteroids on lung → ↓ surfactant)

 ¬ esp. Phosphatidyl glycerol
 - Hypoglycemia (due to the 1ed fetal insulin production)
 - Hypocalcemia & hypomagnesemia → tetany
- 3 ** Polycythemia \rightarrow d.t. chronic hypoxia \rightarrow erythropoietin
 - , Hyperbilirubinemia $d.t. \rightarrow prematurity$, polycythemia, oxytocin
 - . Hyperviscosity syndrome \rightarrow renal vein thrombosis
- Birth trauma \rightarrow shoulder dystocia (wider than the head) + infection CFMF \rightarrow the most common cause (40%) of PNMR \checkmark ^a PNMR (4–10 %) d.t. all the above \checkmark causes

nvestigations

- DM is diagnosed for the 1st time in pregnancy in 90% of cases
- □ History (present, past, family, obstetric) may be suggestive but investigations are a must.....(as symptoms are query: pppp)

O Screening

- 1) Glucose in urine (the worst) X
 - * Done by: Benedict / Fehling test....or glucose strips (easier)
 - * Inaccurate as glucosuria could be due to
 - Renal glucosuria
 - Alimentary glucosuria
 - Lactosuria (some breast lactose → escape in urine)
- 2) Fasting blood glueose (N: <105 mg/dl).... ideal 60-90
- 3) One hour postprandial (N: <140 mg/dl) the best
- 4) Two hours postprandial (N: < 120 mg/dl)
- 5) Random blood sugar (N: < 200 mg/dl)
 - The best screening test is 1hr-PPS //
 (Glucola test -50gm-)
 - > Time
 - For all patients (low-tisk) at 24–28 wks (universal screening i.e. done for all (*) pregnant women without C/O)
 - For high-risk groups → at booking (1st antenatal visit):
 - 1. Maternal obesity or age > 35 yrs
 - 2. Chronic hypertension / renal disease
 - 3. Positive family history
 - 4 History of "
 - . GDM / IGT
 - , Fetal macrosomia
 - . Idiopathic polyhdramnios
 - . CFMF
 - . Unexplained IUFD
 - Result
 - $< 140 \text{ mg}^{-3} \text{ i} \rightarrow \text{no further investigations or ttt}$
 - 140 mg ³ → 3 hr GTT

Modified Priscilla White classification

	Onset Duration Complications			
A	Asymptomatic but with diabetic GTTA ₁ : FBS <105 (→ diet)A ₂ : FBS > 105 (→ insulin)			GDM
B	>20 yearsor	0-9 years	No	IDDM without
C	10-19 yearsor	10-19 years	No	EOD
D	<10 yearsor	>20 years	Vascular / BGR	
F R H	D.M. + Nephropathy D.M. + Proliferative Retinopathy D.M. + Ischemic Heart disease D.M. + Renal Transplantation			IDDM with EOD

National Diabetes Data group classification (1979)

Type I: IDDM

Type 2: NIDDM

• Type 3: DGM

Type 4: IGT

@ Confirmatory (GTT): glucose challenge

(Modified O'Sullivan test)

- Daily 150 gm CHO diet is allowed for 3 days (with no smoking ")
- FBS is determined (after overnight fasting of 8-14 hrs)
- Then give 50 (UK), 75 (WHO), 100 / (USA) gm glucose in 400 ml water
- Readings are taken hourly for the next 3 hrs & a curve is drawn

	Normal Blood Flasma	Renal	Alimentary	D.M.
FBS	< 90 < 105	All	N	At least 2
1-hr	< 165 < 190	readings	> 180 mg %	readings are
2-hr	< 145 < 165	are as	N	>N
3-hr	< 125 < 145	normal	N	
Urine		+ve only	+ve at	+ve at
sugar	All are negative	at peak	1-hr peak	any time

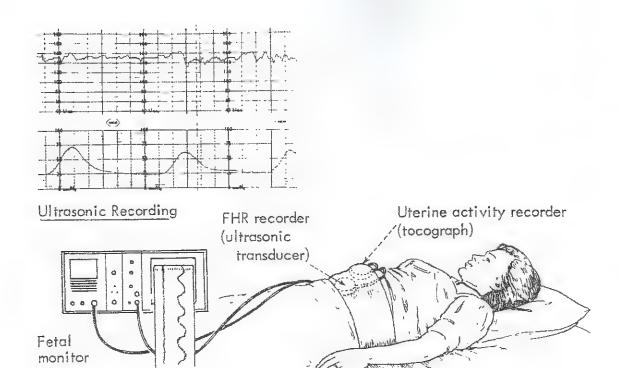
- * Single abnormal value is called → impaired glucose tolerance (IGT)
- * Normal values are to be repeated at 3rd trimester in the high-risk group /
- * If glucosuria found on 2 separate occasions during ANC \rightarrow GTT *

O Investigations for control

- * Level of glycosylated Hb (HbA_{1c})
 - This indicates the control over the previous 2–3 months (N = 5-8%)
 - Above 10% → poor control
 - Above 12% in early pregnancy → ↑% of CFMF *
- * Glycosylated serum proteins, fructosamine (reflects previous control)

Investigations for complications

- * On pregnancy
 - Urine analysis \rightarrow UTI
 - Albuminuria \rightarrow PET
- * Of DM
 - Kidney function tests to detect renal affection
 - Fundus examination
 - . Background retinopathy → not dangerous
 - . Proliferative retinopathy \rightarrow very serious & may lead to blindness \rightarrow refer to laser therapy \rightarrow if still progressive \rightarrow consider TOP



Management

O Preconceptional care

- □ Postpone pregnancy...till good DM control (as evidenced by HbA_{1c})
- Advice against pregnancy if
 - HbA_{1c} > 12% (high risk of CFMF) } indications
 - Marked renal affection is present } of therapeutic
 - Progressive proliferative retinopathy } TOP
- Oral hypoglycemic drugs are not used
 - \(\) they cross the placenta: CFMF + ↑ fetal hyperinsulinism

Antenatal care

- \Box Time \rightarrow 2 wks (3 in GDM) till 32 wks, then weekly
- □ Place → a specialized antenatal clinic (obstetrician, physician, dietitian)
- □ Aim → * control of DM & prevention of its progression

 * early detection & management of comp. (general / obstetric: M&F)
- □ Control 'STRICT' ✓
 - Diet
 - ▶ Sufficient alone only in mild cases (GDM A₁, IGT)
 - . Give CHO (50% = 200-250 gm) fats (30%) proteins (20%) $^{\text{m}}$
 - . Carbohydrates should not be in the sugar form (rapidly absorbed)
 - . Average 1800–2400 Kcal/d ± 300 Cal in 3rd trimester **
 - . Total calories are divided among 3 major meals +3 snacks
 - ▶ Exercise allowed → physical activity should be moderated
 - Diet + insulin
 - ▶ Split schedule system (7 am & 5 pm)...regular + intermediate
 - Indication
 - . GDM A₁ if diet failed: FBS> 105...1hr PPS>140...2hr PPS>120
 - . GDM A2, Class B-T
- □ Investigations
 - Mat. comp: Of DM → renal FT, liver FT, fundus, serial HbA_{1c}
 On preg → screen for PIH, infections (urine, vaginal $C_&S$)
 - ⇒ Fetal surveillance
 - GDM A
 - U/S at 38 weeks to exclude fetal macrosomia
 - CTG & BPP weekly starting from 34 weeks
 - GDM A₂ & IDDM B-T (pregestational IDDM)
 - U/S at .18-20 wks (excludes CFMF)...... \pm MS- α FP
 - . Serially (for macrosomia or IUGR)..... ± Doppler
 - CTG & BPP weekly starting from 32 weeks

Termination of pregnancy

> Time

- □ Diabetics should not be allowed to pass dates >40 wks x
- □ In mild cases under excellent control (GDM class A₁) \$\sim 40 \text{ wks}\$
- □ Insulin requiring diabetics (Class A2, B, C, D)
 - Well controlled, no F/M complications
- 38-40 wks
- Not well controlled: once document maturity
- 37 wks
- ▶ Earlier TOP < maturity if F/M distress occur
- <37 wks
- □ In cases with repeated unexplained IUFD terminate
- 1-2 earlier

Before any elective termination

- ➤ Tests for fetal lung maturity should be done by *amniocentesis*:
 - Shake test (easy)
 - L/S ratio (more specific → widely used)
 - Phosphatidylglycerol → RDS may occur in spite of mature L/S

> Mode

- ♦ Cesarean section: > +
 - * Macrosomia (> 4kg ?!). This is > in GDM
 - . Deposition of glycogen is more at shoulders & fetal liver
 - . The disproportion between fetal head / abdomen → sh. dystocia
 - * Previous history of unexplained IUFD
- * Vaginal \rightarrow by AROM \pm syntocinon (?) + intrapartum fetal monitoring

> Insulin management during TOP

- Keep maternal euglycemia (80–100) → to avoid fetal hypoglycemia
- Before labor stop morning insulin (taken only at bedtime)
- During labor 500cc 5% glucose + 5 units crystalline insulin by drip /5 hrs
- After labor → insulin requirements usually drop immediately
 - If glucose level is $> 200 \rightarrow S.C.$ regular insulin when needed
 - If glucose is persistently > 200 → resume combined regular & NPH

Postpartum care

- □ Care of the newborn.....at the NICU by expert pediatrician
- □ Breast-feeding....encouraged ⁿ (lactation is anti-diabetogenic)
- Contraception
 - COC are contraindicated in those with vascular changes (: use POP)
 - NCD are contraindicated esp. in those with depressed immunity

 - ➤ Threads cut short ± prophylactic antibiotic

Glucose Monitoring and Insulin Dosing During Pregnancy

Insulin Type and Dose Time	Time Impact Seen	Target Glucose Level (mg/dL)
Evening NPH	Fasting	70–90
Morning Humalog	Post breakfast	100-139
Morning NPH	Post lunch	100-139
Evening Humalog	Post dinner	100–139

Instruction for Adjusting Insulin Dosage

- 1. Establish a fasting glucose level between 70–90 mg/dL.
- 2. Only adjust one dosing level at a time.
- 3. Do not change any dosage by more than 20% per day.
- 4. Wait 24h between dosage changes to evaluate the response.

Insulin therapy =

- > Indications:
 - . GDM A₁ if diet failed
 - . GDM A2, Class B-T
- > Drugs used:
 - . Combination of Crystalline (regular / short) } better human + NPH (isophane / intermediate) } e.g. Mixtard
 - . Long acting insulin (protamine zinc) x are not used: poor control
- ➤ Dosage
 - In class B−T → no change in previous dosage (if sugar is controlled)
 - In GDM A₂ give \rightarrow 0.6 u/kg (1st trimester), 0.7 u/kg (2nd), 0.8 u/kg (3rd)
 - The calculated dose is then divided

2

Morning (7 AM)	Evening (5PM)	Assess action at		Urine .
² / ₃ dose	1/3 dose	Morning	®	10 AM
73 dose	+		NPH	5 PM
1/3 crystalline		Evening	®	8 PM
+ ¾ NPH	+ 1/2 NPH		NPH	7 AM

- Urine should be free of glucose: if present → increase the corresponding insulin dose (but gradually!)
- Gluco-meter (capillary blood estimation) is <u>better</u> * used instead of urine for follow up of dosage (d.t. gluco....)
- Check FBS & 1 hr-PPS after each meal until control, then twice weekly

 → then once weekly (1 hr-PPS should be < 140 mg/dl^a)

> In resistant cases

- . May give three times daily injections (NPH at 5 PM is given 1 hr < bed time snack → better control of nocturnal hypoglycemia & FBS)
- . Continuous infusion pump (not superior on injections)
- ➤ <u>Site of injections</u>: given S.C. in abdomen \checkmark (?) \rightarrow arms \rightarrow thigh \rightarrow buttocks Patient is to be taught with variation of the injection site
- The patient should be warned against symptoms of hypoglycemia (headache, sweating, palpitation, hunger, epigastric pain, dizziness)
 & if any of these occur she should take a readily available carbohydrate (candy or biscuit) + ↓ the corresponding insulin dose

KEYPONTS

- Gestational diabetes occurs in 1% to 12% of pregnant women.
- Risk factors for gestational diabetes include
 Hispanic, Asian American, Native American, and
 African American ethnicity, obesity, family history
 of diabetes, and prior pregnancy complicated by
 gestational diabetes, macrosomia, shoulder dystocia, or fetal death.
- All pregnant women should be screened for diabetes between weeks 24 and 28. High-risk women should also be screened at their first prenatal visit.
- Fetal complications of gestational diabetes include macrosomia, shoulder dystocia, and neonatal hypoglycemia.
- Pregnancy management should include frequent health care visits, thorough patient education, American Diabetic Association diet, glucose monitoring, fetal monitoring, and insulin or an oral hypoglycemic agent as indicated.
- Patients should generally be induced between 39 and 40 weeks gestation. Intrapartum insulin and dextrose are used to maintain tight control during delivery. Cesarean section is offered if fetal weight is over 4500 g.

KEY POINTS

- Maternal complications of diabetes during pregnancy include hyperglycemia, hypoglycemia, urinary tract infection, worsening renal disease, hypertension, and retinopathy.
- Fetal complications of diabetes during pregnancy include spontaneous abortion, congenital anomalies, macrosomia, IUGR, neonatal hypoglycemia, respiratory distress syndrome, and perinatal death.
- Pregnancy management is optimized by a preconceptional visit, early prenatal care, thorough patient education, tight glucose monitoring and management with insulin, fetal monitoring, and thoughtful plan for delivery.
- Motivated type 1 diabetics can usually maintain tighter control on an insulin pump. Management in labor and delivery usually requires an insulin drip; however, insulin requirements decrease dramatically postpartum.

- Important points -

Gestational DM @

- * CHO intolerance recognized for the 1st time during pregnancy & disappears after pregnancy (whether insulin is used or not for ttt)
- Screening for GDM should be performed between 24-28 wks

Types	Management	Termination		
Low risk / A ₁	diet control	Left till term (never past-dates)		
High risk / A ₂	diet + insulin	Managed as IDDM		

- More liable to macrosomic fetus (IDDM → CFMF & IUGR)
- Postpartum Consequences
 - Risk of type II DM (50% may develop overt DM within 20 yrs) **
 - Recurrence of GDM (reported in 3/3 of cases esp in obese women)

Glucose intolerance

- If there is only one abnormal value in the 3 hr-GTT
- ³ These patients are still at risk for \rightarrow macrosomia & PIH
- * TTT → only diet control but recheck FBS & 1hr-PPS every 2 weeks

Q. Types of diabetic comas

- Diabetic ketoacidosis
- Hypoglycemic coma
- Hyperglycemic hyperosmolar non-ketotic
- Uremic & cerebrovascular strokes
- Q. Types of insulin Beef / pork / Human ✓ ✓ (mixtard) better

	≈ Onset	≈ Peak	\approx Duration
Short acting (regular, semilente)	½ hour	3-4	6-8
Intermediate (NPH, lente)	2	8-12	16-24
Long acting (PZI, ultralente)	8	12-16	24-32

NPH is the neutral protamine of Hagerden

 \bigcirc . Types of ketone bodies \rightarrow Acetone, Aceto-acetic, β -hydroxy-byuteric acid

Somogyi chenomena

- Increased morning FBS + nightmares.
- Explained by nocturnal hypoglycemia followed by exaggerated counter-regulatory mechanisms → ↑ FBS
- TTT $\rightarrow \downarrow$ NPH of 5-PM

Dawn phenomena

- Increased morning FBS + absent nocturnal hypoglycemia
- TTT \rightarrow \uparrow NPH of 5-PM

Heart disease with preg. (1%)



Types

- Rheumatic \rightarrow 93% (MAT esp \rightarrow MS)
- Congenital → 7% (> in developed countries)
- \rightarrow 1% (e.g. IHD, arrhythmias, cardiomyopathy)

Classification

American New York Heart Association [NYHA] divided HD into 4 grades (according to the functional capacity of the heart):

CLASS I Organic heart disease but with **no** limitation of physical activity

CLASS II Some limitation of the ordinary activity in the form of dyspnea, fatigue, palpitation, pain. $A \rightarrow mild$ limitation . B \rightarrow *moderate* limitation

CLASS III Marked limitation of physical activity as it will lead to dyspnea on less than ordinary activity

CLASS IV They are in heart failure with dyspnea even at rest

Clinical Picture

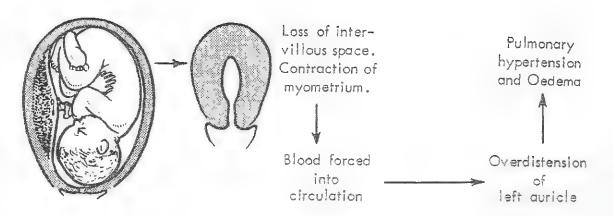
History

Personal

- Name / Age / Marital status / Parity
- Address: ---- RHD is > in damp non-sunny area
- Occupation: ---- may need advice against marked physical effort
- Special habits: --- must stop smoking

Complaint & HPI:

- 1] $\underline{PVC} \rightarrow \text{dyspnea}$, orthopnea, PND, cough, expectoration, hemoptysis
- 2] <u>SVC</u> → engorged neck veins, rt hypochondrial pain, ascites, LL edema
- 3] Rheumatic activity > carditis, arthritis, chorea gravidarum, SC nod, erythema
- 4] *IEC* → fever, symptoms of HF, CNS sympt., hypochondrial pain, haematuria
- $5] Arrhythmia \rightarrow palpitation$
- 6] *Cyanotic heart disease* → cyanosis (malar flush in pregnancy)
- 7] <u>Ischemia</u> → anginal pain
- Menstrual history → for dating
- ➤ Obstetric history → previous HF in pregnancy
- Past history
 - Medical → rheumatic fever, duration of heart disease, attacks of failure
 - Surgical → valve replacement
 - Drugs → anti-failure / anti-coagulant



H.F. immediately after labor

Examination

- * Peripheral & Normally we could see
 - Signs of SVC
 - Neck veins \rightarrow not reliable d.t. \uparrow^{ed} blood volume
 - Edema \rightarrow may occur due to (pregnancy or PET)
 - Enlarged liver \rightarrow may be difficult to palpate due to large uterus
 - signs of hyper-dynamic circulation e.g. H₂O hummer pulse, cap. pulsation
- ❖ Central ☞ Normally we could hear ⇒
 - Splitting of the 1st sound
 - Appearance of the 3rd sound
 - Soft systolic murmurs (< 2/6)
 - Shift of apex beat from 5th to 4th intercostal space

Complications $\Phi\Phi$

M Effects of pregnancy → HD

- 1] Deterioration by one clinical grade due to:
 - *Blood volume 140-50%
 - * COP \uparrow 30% (due to \uparrow blood volume + \downarrow P.R.)
 - = (Heart rate \uparrow 10-15 b/m) X (Stroke volume \uparrow)
- 2] Heart failure may occur in
 - * Pregnancy → at 30–34 wks (max. ↑ in COP & blood volume)
 - * <u>Labor</u> \rightarrow pain + uterine contraction \rightarrow VR to heart \rightarrow COP (2nd stage > 1st stage d.t. more bearing down)
 - * 3rd stage → return of the blood in the uteroplacental circulation (500-700) to G.circulation (after placental separation)
- 3] RHD: . IEC after any procedure (esp in puerperium)
 - . Recurrence of rheumatic activity is rare (but serious if occurred)
- 4] Liability to arrhythmia
- 5] Liability to more cyanosis in cyanotic heart disease
- 6] *Liability to the thromboembolic* complications (due to \(\frac{1}{2}\) stasis)

& Effects of HD → pregnancy

- ☆ Maternal
 - Polyhdraminos (d.t. congestion)
 - PTL (d.t. soft cervix)
 - PPhge (hypoxia + ergometrine contraindicated)
- 六 Fetal
 - Abortion & CFMF } Low COP → chronic hypoxia
 - IUGR & IUFD) esp in cyanotic heart disease

Some give Heparin all through		
* Advantage	* Disadvantage	
 Doesn't cross the placenta Short acting (2-4 hrs) Have antidote protamine sulfate slowly IV 	 Overdose ⇒ bleeding tendency Long use Thrombocytopenia Osteoporosis (∴use calciparine) 	

Some give Oral anticoagulant all through as

- The risk of *over* or *under* control by heparin is more serious ⁿ than the minimal recorded risk of fetal affection d.t. OAC
- FFP rapidly reverses action of OAC (if bleeding occurs)
- Also, there is antidote \rightarrow Vit K (for both mother & fetus)
- NB:- Dindivan is contraindicated during lactation "

Investigations

- 1. X-ray ?! (+ abdominal shield) \rightarrow cardiomegally
- 2. ECG / Echocardiography
- 3. Rheumatic fever \rightarrow ESR, CRP, A-SOT

Treatment

Preconceptional control

- ightharpoonup Pregnancy is contraindicated in: $\Phi\Phi$
 - Class III & IV
 - Cyanotic heart disease as Eisenmenger syndrome
 - Severe Aortic stenosis or 1^{ry} P. hypertension (d.t. limited COP)
 - History of HF in previous pregnancy
 - Rheumatic activity / IEC in the past 2 years
- If they become pregnant, therapeutic TOP is better done in 1st trimester. After 14 wks inducing abortion is more *hazardous* \$\frac{1}{2}\$ than continuing pregnancy

Antenatal care

Done in

a specialized antenatal clinic (obstetrician, cardiologist)

Done for

control of HD & early detection & management of comp

Done every

2 wks till 32 wks, then weekly

Done by

- \neg Rest \rightarrow some hospitalize at [30–34 wks] then to plan labor [36–37 wks]
- □ **Diet** → salt restriction
- □ Drugs →
 - . Avoid anemia / infections (esp. resp tract) \rightarrow ppf to heart failure
 - . Long acting (benzathine) penicillin 1.2 million IU/month ✓
 - . Class III & IV → digitalis, diuretics, aminophylline
 - . Valve replacement with pregnancy:-
 - ☆ Warfarin (5mg) or Phenindione (50mg)
 - ☆ BUT use Heparin (5000 IU/S.C./8hrs) during

1st trimester (as warfarin ↑ CFMF as microcephaly, optic atrophy, chondrodysplasia punctata + ↑ fetal hge)
 2-3 weeks before delivery ⇒ shift back to heparin

At onset of labor \Leftrightarrow stop heparin

After labor (6-12 hrs) = give OAC + heparin (till OAC acts)

3 days later ⇒ stop heparin.....then continue only by OAC

Regimens for endocarditis prophylaxis during labor and delivery

Low risk regimen Amoxicillin, 3 g p.o. 1h before procedure or at onset of labor

Repeat 1.5 g p.o q.6 h until after delivery

Standard

Ampicillin, 2 g i.v. plus gentamicin, 1.5 mg/kg i.v.

regimen

(do not exceed 80 mg) 30 min before procedure or

at onset of labor. Repeat above q.8 h until after delivery

Penicillin-allergic Substitute vancomycin, 1 g i.v. over 1 h q.12 h for ampicillin

standard regimen

History

Etienne-Louis Arthur Fallot (1850–1911) was a professor of forensic medicine and hygiene in Marseille. He had a reputation as an astute clinician and for accurate careful physical examination.

@ Termination

- ♦ Time
 - Class I & IIleft for smooth spontaneous of labor (no induction?)
 - Class IIIif completed here family → better to terminateif insists on pregnancy → continue in hospital
 - Class IV......control the HF 1st medically, then terminate
- Route
 - > vaginal ✓✓

1st stage

- Semi-sitting position with no bearing down
- Adequate analgesia
 - * Morphine (10mg) or Pethidine (100mg)
 - * Epidural analgesia
- Intermittent O₂ + antifailure ttt if needed
- Close observation [P-BP-Temp] + FHS + Uterine contraction
- Prophylactic antibiotics (GBS ✓) & delay AROM as possible
 - * 2 g ampicillin + gentamycin 1.5 mg/Kg
 - * given 1 hour before placental separation
 - * Ampicillin is repeated once after 8 hours

2nd stage

- Usually easy (small baby + soft cervix)
- Shorten 2nd stage & avoid bearing down by low forceps or ventouse

3rd stage

- Avoid ergot IV (↑ heart load due to VC + uterine contraction)
- Lasix may be given ✓ (if heart failure)
- Guard against PPhge (may give 1/4 mg IM)

> cesarean section == +

- 1] Aortic stenosis (post-stenotic dilatation may rupture during bearing)
- 2] I'' pulmonary hypertension
- 3] Eisenmenger syndrome
- 4] Marfan syndrome (if having dissecting aortic aneurysm)

Postpartum care

- * Breast-feeding allowed except in severe cases (class III & IV)
- * Advise for contraception > Mechanical methods or Sterilization
 - o IUCD → ascending infection (if used: aseptic technique + proph. abtc)
 - \circ COC \rightarrow thrombosis (if used: POP)

SOME IMPORTANT CARDIAC CONDITIONS

1] Mitral Valve Prolapse

Pathology: Myxomatous degeneration of one or both of mitral valve leaflets → prolapse into left atrium during systole

C/P: - Asymptomatic mainly

- May → palpitation, dyspnea, chest pain, syncope

Treatment during labor: Controversial → only inderal
Most don't give antibiotics except if associated with MR

2] Peripartum Cardiomyopathy

Definition: Dilated cardiomyopathy → HF in 3rd trimester / puerperium

Incidence: 1/1.500 - 1/4.000

Etiology: - Unknown

- May be \rightarrow viral infection, autoimmune

- Pdf → PET, twins, genetic predisposition

Prognosis: (Fatal). Mortality is 25-50%

. Mortality in next pregnancy is 80%

Treatment

- 1. Digitalis, hospitalization
- 2. ↓ afterload (Hydralazine), ↓ preload (diuretics)
- 3. Heparin (risk of thrombus formation in dilated heart)

3] Coarctation of Aorta

Definition

- Hypertension only in upper limbs
- Normal / low pressure in lower limbs
- May be confined only to left arm (coarctation of left subclavian)

Route of termination

- Vaginal delivery allowed
- C.S. only in other obstetric indications

4] Marfan Syndrome

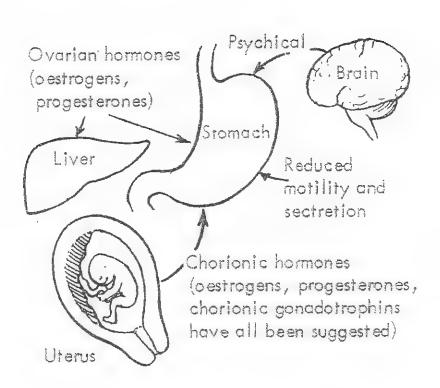
Etiology → defective CT

Clinical picture

- Mitral valve prolapse / incompetence
- Aortic dissection (intimal tear) → acute chest pain + shock

5] Surgery in pregnancy

Tight MS (< 1cm) Balloon catheterization may be done (2nd trimester) **Valve replacement** is contraindicated (hear lung machine + anticoagulation)



Vomiting with pred

Emesis gravidarum (morning sickness)

- > Definition
 - N_&V in the 1st trimester (max 6th 12th week)
 - It doesn't affect the general condition
- > Incidence = very common (80%) esp in PG, esp in the morning
- > Etiology = unknown
- > Management
 - Reassurance → it disappears spontaneously
 - Small frequent meals →. Better dry CHO meals
 - . Avoid immediate recumbency after meals
 - . Fe therapy is temporarily stopped (nauseating)
 - If not responding → antiemetics

P Hyperemesis gravidarum Z

- > Definition
 - Severe vomiting to a degree that
- } pernicious
 } vomiting of preg
- Affects the general condition
 Incidence = 0.1−1 %
- > Etiology = theories
 - Psychological: as it
 - . Start only after knowing that she is pregnant
 - . Vomiting only infront of her husband $\ensuremath{\mathfrak{D}} \to \mathsf{more}$ in neurotic females
 - Hermonal
 - . ↑ HCG (as in V.mole & twins)
 - . ↑ T₃, T₄transient....no need for ttt
 - . L Corticosteroids
 - Allergie → against CL of pregnancy, sex steroids
 - Deficiency (esp Vit B₁ & B₆)
- > Pathology = vomiting)
 - Starvation → dehydration → starvation ketosis & elect. imbalance
 - Liver fatty change & centrilobular necrosis
 - Kidney tubular necrosis
 - Heart brown atrophy
 - Brain petechial hge & congestion
 - Ratina hge, optic neuritis, detachment

Clinical Picture

- Excessive vomiting (allover the day & not related to meals)
- Dehydration → . ↓BPr, ↑pulse, ↑temp...... oliguria, constipation . ↓weight, sunken eyes + jaundice, dry inelastic skin
- $CNS \rightarrow .$ Peripheral neuritis
 - . Wernicke's encephalopathy d.t. Vit B₁ def

Investigations

- - \Rightarrow CBC $\rightarrow \uparrow$ Hct.... \downarrow (Na, K, cl)
- Etiology \Rightarrow U/S (Twins, V.M.) + T_2 , T_4
- Complication
 Fundus, Liver + Renal function tests

Treatment

[1] Hospitalization reassurance (& isolation!)

[2] Diet

- NPO + IV fluids (till 48 hrs after vomiting stops)
- Then restart gradually by clear liquids + CHO meals (no fats, spices)
- If failed \rightarrow TPN + thiamine (B₁)

[3] Drugs

- Sedatives → phenothiazines (chlorpromazine)
- Antihistaminic → promethazine (phenergan)
- Antiemetics → . metoclopramide (primperan), Cortigen B₆
 . navodoxine, motilium (domperidone)
- In resistant cases \rightarrow Zofran (ondansteron: 5–HT blocker) \pm steroids

[4] Observation for

- Vomiting: → frequency
- Vital data: \rightarrow BPr, P, T
- Urine analysis → daily
- Organ function tests + Fundus → weekly

[5] TOP

** Indication

Deterioration of general condition in spite of ttt [P > 100, T > 38]Deterioration of organ affection [renal / hepatic / CNS / retina]

** Methods

- < 14 weeks -> Suction evacuation or D&C
- $> 14 \text{ weeks} \rightarrow \text{may end in } \underline{\text{HYSTEROTOMY}} \dots \text{why?}$

Complications of cholestasis of pregnancy

Comments
Comments
Due to malabsorption of vitamin K
2-4%, with risk increasing with gestation
40% will deliver before 37 weeks
Meconium-stained amniotic fluid likely
Due to maternal absorption of vitamin K

---- Extras ----

DD of vomiting in pregnancy

 $\Phi \Rightarrow$

- 1. Morning sickness (emesis gravidarum)
- 2. Disturbed ectopic pregnancy
- 3. Vesicular mole
 - 4......Preeclampsia (severe)
 - 5.....Pyelonephritis
 - 6.....Polyhydramnios
- 7. Gyn. conditions → twisted ov. swelling, red degen. of fibroid
- 8. Medical *conditions* \rightarrow food poisoning, hepatitis
- 9. Surgical *conditions* → appendicitis, cholecystitis, peptic ulcer

DD of Jaundice in Pregnancy



Pregnancy induced	Pregnancy associated
- PIH & HELLP syndrome	- Hemolytic J.
- Severe hyperemesis gravidarum	- Obstructive J.
- Intrahepatic cholestasis of preg	- Hepatocellular (V.H. ✓✓)
- Acute fatty liver of pregnancy	- Drugs

• Intrahepatic cholestasis of pregnancy 🚓

- *Incidence o the commonest liver disorder <u>UNIQUE</u> to preg. (esp. in 3rd trim)
- *C/P \rightarrow mild Jaundice, pruritus + \uparrow in Bile acids are <u>DIAGNOSTIC</u>
- *Comp. \rightarrow PTL & IUFD
- *Investig. \rightarrow . \uparrow ^{ed} (alkaline phosphatase, Bilirubin –direct–, SGOT & SGPT)
 - . Prolonged prothrombin time
- *Treatment
 - a mother . cholestyramine 4gm 1x4 ± Phenobarbitone for pruritus
 - . Vit K 10 mg to improve the PT
 - . Corticosteroids may help
 - □ fetus → TOP once mature → MARKED improvement within 2 days
- **Prognosis → avoid use of COC to avoid recurrence (70%)

Acute fatty liver of pregnancy (rare)

- *C/P symptoms like viral hepatitis \rightarrow fever, N_&V, Jaundice, upper abdominal pain
 - Then \rightarrow symptoms similar to PET \rightarrow then acute LCF
- *Complications → high MMR, PNMR
- *Treatment → TOP + liver support

Urinary tract dis. in preg.



[1] Asymptomatic (silent) Bacteriuria 🖄

Definition (4-7%)

- Presence of >100.000 organisms of a single colony /ml urine
- In absence of any symptoms or pus cells

Effects on pregnancy $\rightarrow \uparrow^{ed}$ liability to:

- Acute Pyelonephritis (in 25% of cases)
- Anemia
- PIH & IUGR
- PROM & PTL

Diagnosis

- No symptoms ^a ∴ screening tests MUST BE DONE in 1st visit Colony count (by clean catch technique: MSU)

Treatment

- 1- Broad spectrum antibiotic (of high urinary concentrations) for 7–10 d [Ampicillin / Cephalosporins / Nitrofurantoin]
- 2- If failed \rightarrow antibiotic according to $C_{\&}S$
- 3- Urine C&S is then repeated each trimester "

[2] Acute Pyelitis & Pyelonephritis

Definition (1%) → acute infection of renal pelvis & interstitial tissues

Predisposing factors "

- Short urethra
- Asymptomatic bacteriuria

- ⇒ ↑ % of infection
- Atony of the urster (Pr. effect)
- ascending infection
- Pressure on ureter (esp. Rt.) at pelvic brim
- after 2nd trimester
- Catheterization (urinary tract is until) = after labor

Bacteriology

- > Organisms
 - E-coli \rightarrow the most common \checkmark (80%)^a
 - Others → Gram +ve : staph / strept
 Gram -ve : Klebsiella / Proteus
 Infections
- > Route of infection
 - Ascending infection.....along the lumen or periureteric lymphatics.
 - Lymphatic spread......from neighboring colon
 - Blood borne from.....a septic focus (rare)

Clinical picture

* Sumptoms

- General → FAHMR + vomiting (sudden onset)
- Local → severe loin pain, dysuria, haematuria / pyuria, frequency

* Signs

- General → high fever, tachycardia
- local → tenderness & rigidity in loins

Investigations

- Urine analysis → Pus cells, RBCs
 - Acidic with fishy odour in E-coli
 - Urine C&S (esp in recurrent cases)
- Renal function tests → urea & creatinine may be affected
- Blood picture $\rightarrow \uparrow$ ESR, TLC, CRP

Differential diagnosis acute pain in pregnancy

Complications

- Chronicity.......Pyonephrosis......Perinephric abscess
- Uremia......Septicemia.....Septic shock
- PROM & PTL
- Recurrence in next pregnancy (20%)

Treatment

Ceneral

- Hospitalization
- Ample fluids (oral or IV) + Analgesics + Antipyretics

→ Antibiotics → at least for 2 wks (best according to C&S)

- Most commonly used \rightarrow . Ampicillin 500 mg 1x4 . Cephalosporins 500-1000 mg 1x4 1x4
 - . Nitrofurantoin 100 mg
- If no response within 72 hrs \rightarrow repeat $C_{\&}S$
- In resistant cases \rightarrow postpartum IVP, U/S \rightarrow may reveal stones, congenital anomalies (esp if there is hematuria, hypertension)

Surgery

- If ureter is blocked → ureteric catheter (pig-tail)
- If failed → nephrostomy
- \hookrightarrow TOP \Longrightarrow If treatment fails to control the condition

[3] Acute renal failure in preg &

- > Definition rapidly progressive azotemia
- > Etiology 1
 - Pre-renal failure (hypovolemia: accidental hge or hyperemesis Gr.)
 Renal
 - Sepsis (e.g. septic abortion)
 - PET, HELLP syndrome, DIC
 - · Hepato-renal → acute fatty liver in pregnancy
- > Two types

ACUTE TUBULAR NECROSIS (reversible)

BILATERAL CORTICAL NECROSIS (rare & more worse)

[4] Chronic renal dis. with preg &

- > Effect of pregnancy --> renal disease
 - Mild (creatinine < 1.4 mg%).....no change in renal function
 - Moderate (creatinine 1.4 2.5 mg%)....deterioration of renal function
 - Severely (creatinine > 2.5 mg%)..... usually don't get pregnant
- > Effect of renal disease -> pregnance
 - t Effects
 - Superimposed PE ✓ (the most serious) ± accidental haemorrhage
 - Chronic anemia
 - Abortion / IUGR / PTL / IUFD
 - a Prognosis depends on
 - Development of hypertension
 - Development of proteinuria
 - Degree of renal impairment
 - Type of renal disease (diffuse & proliferative GN are worse)
- > Treatment
 - Mild = conserve under strict observation

(monitor renal function + antihypertensives

Severe = terminate or if deteriorating

advise sterilization or do it with termination

NB

- ? Pregnancy is possible on <u>dialysis</u> (but chronic anemia is the major problem)
- Pregnancy is possible after <u>renal transplantation</u>
- Delivery is better vaginal (even after renal transplantation, as kid. is in.....)

Anemia with prequancy



Definition = in the amount of circulating haemoglobin (N= 12-16 gm%)

Normal changes in Holingistande

- ³ Physiological Anemia (haemodilution) due to
 - RBC volume ↑ by 20–30 % but
 - Plasma volume ↑ by 40–50 %
- ^a Lower limit is 11 gm/dl (Hct < 33%). Below this → PATHOLOGICAL ANEMIA *

Classification:

atsiogomsorby [].	1) Iron deficiency anemia ✓ (the commonest dis) [*] 2) Megaloblastic anemia . Folic acid ↓ (common) [*] . Vit. B ₁₂ ↓ (rare) [*]
2] Aplastic	Bone marrow failure by . Infections (viruses) . Drugs (chloramphenicol) . Malignancy (leukemia)
3) Haemolytic	 Intra-capsular . Hb → haemoglobinopathies Enzyme → G₆PD Membrane → spherocytosis Extra-capsular → infection, drugs, hypersplenism
4Haemonnage	Acute (APHge, PPHge) or Chronic (e.g. piles)

- _ Microsytts 1111 30 f. iron def anemia, thalassemia
- : Marrosyre 110 1100 sickle cell anemia
- a Marrayi'e and vit B₁₂, folate def

Complications

= Eifect of pregnanc (worsened; d.t. ↑ Fe & vitamin demand)
= Lifect of pregnanc (worsened; d.t. ↑ Fe & vitamin demand)

Pregnancy	- Abortion / Preterm labor
	- Antepartum hge.
labor	- PPhge: atonic
Puerper um	- P. sepsis / Subinvolution
	- Defective lactation

The fetus obtains all its needs of iron & vitamins from the mother by active transport (even if she is anemic). However, there may be \rightarrow . PTL & RDS

.↑ PNMR (d.t. the maternal comp.)

Blood changes in pregnancy

	Non-pregnant	Pregnant
Haemoglobin (g/dl)	12-14	10–12
Red cell count (x1012/l)	4.2	3.7
Haematocrit (venous)	40%	34%
MCV (fi)	75–99	80–103
MCH (pg)	27-31	No change
MCHC (g/dl)	32-36	No change
White cell count (×109/I)	4-11	9–15
Platelets (×109/l)	140-440	100-440
ESR (mm/h)	<10	30–100

ESR, erythrocyte sedimentation rate; MCH, mean corpuscular haemoglobin: MCHC, mean corpuscular haemoglobin: MCV, mean corpuscular volume.

The requirements of elemental iron during pregnancy

Fetus and placenta	500 mg
Red cell increment	500 mg
Postpartum blood loss and 6 months' lactation	360 mg
Total : To the control of the co	1360 mg
Saving from amenorrhoea approximately	360 mg
Net increased demand approximately	1 gram

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Gron Def. Anemia 90% ✓.

1

Pathogenesis

- * Iron requirements in pregnancy are > iron absorption (inspite of its[†])
- * Therefore iron stores in the mother are used to correct the difference
- * If iron stores are already depleted or the mother is anemic iron deficiency anemia occurs or is aggravated

Normal Fe absorption²

- Daily absorption $\rightarrow 10\%$ $-\uparrow^{ed}$ to 20% in preg- of ferous supplied (10 mg/d) \rightarrow Non- preg. (1-2 mg), early preg. (2.5 mg), late preg. (6.5 mg)
- One gram is needed for the whole pregnancy
- > Etiology (Pcf) ₽ nutritional intake or ₽ stores
 1 loss → hge....vomiting...piles...parasitic infestations

Investigations

- Hb % \rightarrow < 11 gm/dl (<10.5 mg % recently)
- Blood picture → hypochromic microcytic, \(\psi_{ed}\) (MCV, MCH, MCHC: <30 g/dl)</p>
- Iron studies: ™
 - S. ferritin ↓ (reflects BM stores) <10 ng/ml (1st abnormal test ✓)
 - Serum iron \downarrow (N. = 60–180 μ g/dl)
 - Bone marrow stores
 - Total iron binding capacity ↑ (reflects ↓ transferrin saturation by 15%) **

> Treatment

♦ Prophylactic

- Eradicate any pdf
- Improve diet + iron supplementation orally (after 1st trimester: N_&V)

 ➤ Iron sulfate / gluconate / fumarate (30–60 mg/day)

* Active

- 1] Oral from 1x3: during or after meals to supply 120-240 mg/day
 - If \rightarrow Oral therapy fails (< 0.3–1 gm rise of Hb / wk)
 - Side effect: N&V, constipation occurred
 - Rapid results are needed, severe cases. Shift to)

2] Parenteral iron

Iron dextran (Imferon) IV or IM X
Iron sorbitol (jectofer, IM) or iron sucrose (cosmofer, IV) ✓
** Side effects: Allergic + . IM → painful + sterile abscess
. IV → thrombophlebitis

3] If severe anemia → Packed RBCs transfusion

. Yesani

DNA replication is affected $\rightarrow \downarrow$ nuclear maturation \rightarrow affection of the 3 cell lines \rightarrow anemia, leukopenia (infections), thrombocytopenia (bl. tendency)

> folic ació deficiency VV

Etiology $\rightarrow \uparrow$ demand e.g. preg + \downarrow intake (no vegetables + \uparrow cooking) Effect — anemia + neural tube defects, cleft lip & palate (fetus) Investigations

- Hb % < 11 gm/dl
- CBC \rightarrow macr. hyperchr. (MCV >100fL), hypersegmented neutrophils $^{\pi}$
- Folic acid level (N = 6-12 μ g/L)
- Bone marrow → megaloblasts

Treatment

 ζ Prophylactic \to . Good diet \pm folic acid supplements (800 μ g/d) . Given also for \to hemolytic anemia, anticonvulsant therapy ζ Active \to 5 mg/day + vit.B₁₂ + <u>Fe</u> (to avoid unmasking ass Fe \downarrow anemia)

> pernicious (addisonian) anemia (vit B, defi

Ethology \rightarrow long term vegetarian diet " + intrinsic factor deficiency Rage to become pregnant (usually infertile)

Effect -- anemia + atrophic gastritis + neurological symptoms

Treatment \rightarrow parenteral vit B₁₂ cyanocobalamin (1000 μ g/3 months/IM/ life)

3

Haemolytic anemia

æ/

> thalassemia

- 1- Thalassemia minor $\rightarrow \downarrow \alpha$ chain synthesis \rightarrow v. mild anemia \rightarrow min. effect on preg
- 2- Thalassemia major $\rightarrow \psi \beta$ chain synthesis \rightarrow severe anemia, rare to become preg

TTT - Repeated blood transfusions + folic acid + No iron

- Splenectomy.....Desferal (iron chelating) to avoid haemosiderosis 🗶

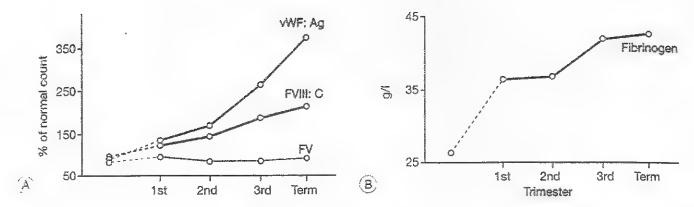
> sickle cell anemia

- 1- Siekle cell trait \rightarrow v. mild anemia \rightarrow min. effect on preg (UTI $^{\alpha}$)
- 2- Siekle cell disease ->
 - Occlusive crisis: obstruction of vessels → infarctions
 - Hemolytic crisis: anemia & jaundice

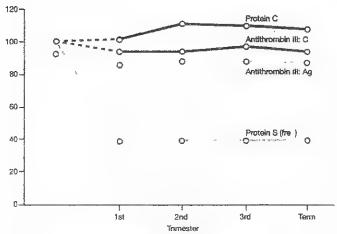
TTT - Prevent occlusive crisis by . Good hydration

. Avoid hypoxia & infections

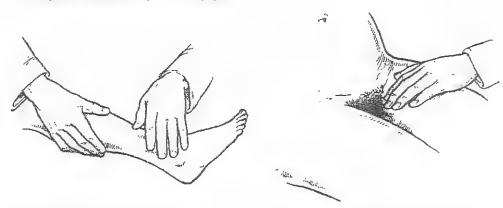
- Repeated blood / exchange transfusion to ↓ level of HbS



The levels of the procoagulants (A) factor VIII, von Willebrand factor and (B) fibrinogen rise in pregnancy. FV, factor V.

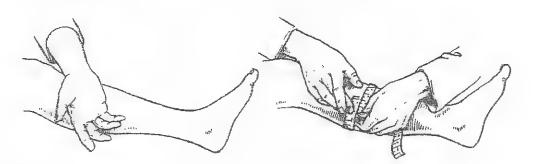


The levels of the anticoagulants antithrombin ill and protein S fall in pregnancy.



Palpation of the calf demonstrates tenderness and oedema.

The femoral vein must also be palpated in the grain.



The affected leg may feel warmer to the back of the hand.

Careful measurement may reveal some swelling compared with the other leg.

Thromboembolic Diseases

Incidence $\Rightarrow 0.5-1\%$

Incidence is \rightarrow equal in both ante- & post-partum periods Untreated DVT \rightarrow PE in 25% of cases with MMR 15%

Treated DVT → PE in 5% of cases with MMR 1%

Factors increasing risk of TED in pregnancy include /

- Delivery operative > vaginal delivery (why??)
- Previous thromboembolism recurrence risk is 15%
- Thrombophilia
 - Factor V Leiden deficiency → ↓ antithrombin III } recurrent
 - Protein C & S deficiency } TED &
 - Anti-phospholipid syndrome } fetal loss
- Changes in Wirehow triad
 - Prolonged immobilization
 - Congestive heart failure, dehydration, sickle cell disease

Diagnosis

- ☆ Symptoms
 - DVT → acute painful swollen leg
 - . More common in the left LL (why..?)
 - . More common in ilio-femoral (more dangerous > calf)
 - $^{\circ}$ PE \rightarrow sudden severe chest pain, dyspnea, cyanosis
- 🕏 Signs
 - Homan's sign (painful dorsiflexion of the ankle)30%
 - Tender hard cord-like may be palpable
 } false +ve
- A Investigations
 - Doppler & colored Doppler
 - They are slightly less accurate in <u>pelvic</u> DVT (MRI may be used)
 - Venography still has better results in calf DVT
 - If PE is obvious → start heparin immediately
 - No need for chest X-ray, ECG, blood gases
 - To confirm PE → perfusion (V/Q) scan, CT, MRI

Complications

राहालगातिक विवास	- 	विवती १५	Orii
MMR =15%. However,	Post-phlebitic syndrome	APS may lead	On both
if recovery occurred	→ valve destruction:	to habitual abortion	1- Mother
→ is usually complete	edema, skin ulceration	(important)	2- Fetus

Management

	- Stepanii	Oral amtionagulant"
Mechanism	activates AT _{III} → ↓ II & 9–12	Vit. K antagonist → ↓ II & 7–10
Curative Control	40.000 u/day IV, for 10 days double APTT	- Better for artificial valves - In VTE, it is used for 4-6
Prophylaxis Control	10.000 units SC, twice daily normal APTT	wks postpartum - PT is kept 2.5 – 3 x (INR) = international normalized ratio
Contraindication	.Active bleeding, active ulcer . CNS aneurysms . Uncontrolled hypertension	Not used in the 1st trimester as it crosses the placenta at the second s
Complications	HaemorrhageThrombocytopeniaOsteoporosis (if used > 3m)	- Embryopathy - Chondrodysplasia Punctate - Fetal Hge: ICHge APHge

Low molecular weight (fractionated) beparin (LMWH)

- ³ Fractionated heparin e.g. Enoxaparin (Clexane) ✓✓, Dalteparin (Fragmin)
- ³ Dose \rightarrow 1 mg /kg /12 hrs (Prophylaxis = 30–40 mg /12 hrs)
- ² Follow up → anti–Xa ✓ (not PTT)

	Figinal approx	पितिस्टिंग्सिंग्सिंग्सिंग
Mode of action ATII	Mainly X	Mainly II
Molecular weight (KD)	4.000 - 6.500	5.000 - 30.000
Bioavailability	90%	30%
Flatt life .	4 hrs (SC or IV)	3 hr (SC)1 hr (IV)
Side effects (hge, thrombo	All are less with LMWH	¤
evionena ostenni	The main disadvantage	is their high

Thromboembolic disease

Risk factors for venous thromboembolism in pregnancy and the puerperium after vaginal delivery

Pre-existing	Alous
11 C-CAISONY	New onset or transient
Previous VTE	Surgical procedure in pregnancy or puerperium, e.g. ERPC
Thrombophilia Congenital Antithrombin deficiency Protein C deficiency Protein S deficiency Factor V Leiden Prothrombin gene variant Acquired (antiphospholipid syndrome) Lupus anticoagulant Anticardiolipin antibodies	Hyperemesis Dehydration Ovarian hyperstimulation syndrome Severe infection e.g. pyelonephritis Immobility (>4 days bed rest) Pre-eclampsia Excessive blood loss Long haul travel Prolonged labour Midcavity instrumental delivery

Age >35 years
Obesity (BMI >30 kg/m²)
Parity >4
Gross varicose veins
Paraplegia
Sickle cell disease

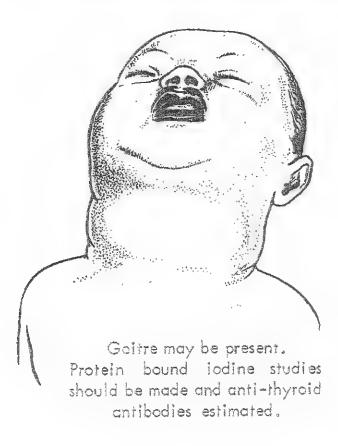
Immobility after delivery

Paraplegia
Sickle cell disease
Inflammatory disorders, e.g. UC/Crohns
Some medical disorders, e.g. nephrotic synd
Myeloproliferative disorders, e.g. ET, PRV

Thromboembolic disease in pregnancy – points to remember

- PTE is the commonest direct cause of death in pregnancy and the puer-perium in the UK.
- Pregnancy is associated with an increased risk of thrombosis.
- The risk of DVT and PTE in pregnancy increases with increasing maternal age and obesity.
- Emergency caesarean section is associated with a greater than 20-fold increase of dying from PTE compared to spontaneous vaginal delivery.
- Objective diagnosis of DVT and PTE is vital.
- Treatment of VTE in pregnancy necessitates larger doses of LMWH and warfarin is avoided.
- Following acute VTE in pregnancy, LMWH must be continued for the rest of the pregnancy and the puerperium.
- Decisions regarding thromboprophylaxis in pregnancy relate to past history of VTE, the presence of detectable thrombophilia and the other identifiable risk factors.
- Women at high risk of recurrent VTE should receive antenatal and postnatal thromboprophylaxis with LMWH.
- LMWH and warfarin are safe to use in lactating mothers.

The papy should be examined carefully after birth.



X-ray may show absence of bone centres.

Thyrotoxicosis

> Complications

- → Effect on thyroid
 - Usually tolerable course during pregnancy
 - Condition may improve & exacerbates after delivery
- → Effect on Pregnancy
 - ♦ Severe hyperthyroidism: usually → anovulation → amen. & infertility
 - ♦ Maternal
 - Spontaneous abortion & PTL
 - PIH & Congestive HF
 - Hyperemesis gravidarum
 - ♦ Fetal
 - IUGR, IUFD
 - Fetal tachycardia, neonatal hyperthyroidism
 - Fetal thyrotoxicosis & goiter d.t. passage of autoantibodies (IgG)

> Treatment

- Propylthiouracil (drug of choice) ✓→ 200-400 mg/d
- Methimazole (carbimazole) → 20-40 mg/d
 - They cross placenta → fetal hypothyroidism & goiter
 - They are not an absolute contraindication to breast-feeding

→ Beta-blocking Agents

- Propranolol (Inderal) 10 mg 1x3
 - . Block the beta-adrenergic receptors
 - . Prevent adrenergic effects of thyrotoxicosis
 - . Block the conversion of $T_4 \rightarrow T_3$
- The aim \rightarrow maintain the lowest possible doses of anti-thyroid drugs
- ⇒ Surgery: Subtotal thyroidectomy is rarely indicated * except:
 - Failed medical ttt
 - Cannot tolerate medical ttt
 - Large goiters with significant tracheal obstruction It does not eliminate the risk of transplacental passage of

LATS and the possibility of fetal & neonatal thyrotoxicosis

 \hookrightarrow Radioactive iodine ablation (I¹³¹) \rightarrow X contraindicated in pregnancy

> Etiology

- o Primary hypothyroidism: (TSH is high)
 - Hashimoto's thyroiditis (autoimmune) ✓✓
 - o Iatrogenic (Radioactive-iodine 131, surgery, antithyroid drugs)
 - Iodine deficiency
- o Secondary hypothyroidism: (TSH is low) Rare, 2" to:
 - O Hypothalamic or pituitary disease, as in Sheehan syndrome?!
 - Chromophobe adenoma of pituitary gland

> Complications

- * Maternal → abortion, PIH, & abruptio placentae, heart failure " "
- * Fetal →. IUGR & IUFD
 - . Congenital hypothyroidism (\rightarrow obstructed labor) occurs in:
 - RAI therapy for thyrotoxicosis 🗸
 - Rarely in hypothyroidism

Investigations

- Low serum T₃RU
- 1 Thyroid antibodies (antimicrosomal, antithyroglobulin) in Hashimoto
 - TSH is low in 2^{ry} hypothyroidism

> Treatment

Replacement therapy

- L-thyroxine (T₄) 0.05–0.10 mg /day converted in body to T₃
- Breast-feeding is not contraindicated

Thyroid function in pregnancy

- o Increased due to
 - ↑^{ed} production of TSH
 - 1^{ed} production of thyrotropin by the placenta
 - The thyroid stimulating effect of β -HCG
- Leading to $\rightarrow \uparrow$ total serum $T_3 \& T_4 \& \downarrow T_3 RU$ However $\rightarrow TBG$ is also \uparrow^{ed}
- o Thus →
 - Free T₃ & T₄ remain normal
 - TSH remain normal

Respiratory disorders



O Breathlessness (dyspnea)

- ▶ Physiological → 50% of normal preg (mechanical & prog effect)
- ➤ Pathological → don't forget pulmonary embolism (acute dyspnea)

9 Maternal Smoking

- > Tobacco smoke (3800 constituents)
 - Nicotine → vasoconstriction
 - $CO \rightarrow$ combines with fetal Hb \rightarrow carboxyhemoglobin \rightarrow fetal hypoxia
 - Benzopyrene → mutagenic & carcinogenic

> Effect of smoking on pregnancy

- IUGR ✓ Neonates are ± 200 g lighter than non-smokers

 Effect is dose related (number of cigarettes/day)
- o ↑²¹ PNMR → including sudden infant death syndrome
- Spontaneous → abortion, PTL, PROM
- o APHge → placental abruption, placenta previa

Sronchial Asthma (1%)

- Complications
 - Effect of pregnancy => asthma: no effect on frequency or severity
 - Effect of asthma = pregnancy: HTN, IUGR, PTL
- > Management
 - Regular medications are CONTINUED (not terratogenic)
 - · Inhalation is better than oral agents
 - . Glucocorticoids (Betamethasone), Disodium cromoglycate & ipratropium "
 - , β_2 agonists, Theophyllines (aminophylline) x
 - · Asthma exacerbation is not an indication for elective delivery
 - Hydrocortisone is given during labor (300 mg/12 hrs)
 - Avoid the following drugs . Prostaglandin $F_{2\alpha}$ & E_2 analogues (misoprostol)
 - . Methergine, Pethidine (not a problem in practice)

4 Surgery in pregnancy > 4

	O Acute Appendicitis *	Ø Acute cholecystitis	Ovarian torsion or Ruptured CL
Incidence	Commonest 1/1500 🛩 "	2 nd common 1 /4000	uncommon
Pdf	Unknown	Relaxation of gall bladder (d.t. progest.)	Induction of ovulation
Diagnosis	difficult *	easier	↑ level of suspicion
C/P	Pain m.b. somewhat upwards. This depends on gest. age	Same like the non- pregnant	Acute unitateral pain ± N _{&} V ± Adenexal swelling
Invest.	TLC is normally ↑ed in preg	Bilirubin, amylase Upper abd. U/S	U/S is essential for all (for differentiation)
Complicat.	Rupture, perforation, peritonitisfetus → abortion, PTL		→ abortion, PTL
Treatment	Laparotomy (its site depends on gest. age) e.g. Ratherford extension	* Medical ttt (Fluids, NPO, A, A, A) * If failed (25%) or complicated → surgery	Look Cancer Ovary

Surgery during pregnancy 🗸

Best time is atmid-trimester (why?)

Before surgery......document fetal life \pm CTG

During surgery.....tocolytic infusion, minimal uterine manipulations

After surgery

Antibiotics

Tocolytics, Profenid suppository ± progesterone

Reassess fetal viability

* Pain in presnancy & 🛎 *

9	Pregnancy		
☆ Early		Abortion, ectopic, incarcerated gravid RVF uterus	
	☆ Late	Accidental hge, rupture uterus, acute fatty liver, acute polyhdramnio	
对 Masses		- Complicated ovarian (ruptured CL cyst or TL cyst of V.mole)	
		- Complicated fibroid (red degeneration)	
2	Urinary	Cystitis, pyelonephritis, stones (renal colic)	
8	GIT	Gastroenteritis, viral hepatitis, food poisoning	
0	Surgical	Acute appendicitis, acute cholecystitis, perforated DU	
9	Medical	DKA, sickle cell crisis, acute porphyria, mesenteric vasc. occlusion	